

# Public Health Assessment

**Initial - Public Comment Release**

**ORLANDO GASIFICATION PLANT**

**ORLANDO, ORANGE COUNTY, FLORIDA**

**EPA FACILITY ID: FLD984169235**

**Prepared by the  
Florida Department of Health**

**SEPTEMBER 13, 2016**

**COMMENT PERIOD ENDS: NOVEMBER 14, 2016**

Prepared under a Cooperative Agreement with the  
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES  
Agency for Toxic Substances and Disease Registry  
Division of Community Health Investigations  
Atlanta, Georgia 30333

## THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

This Public Health Assessment-Public Comment Release was prepared by ATSDR pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA or Superfund) section 104 (i)(6) (42 U.S.C. 9604 (i)(6)), and in accordance with our implementing regulations (42 C.F.R. Part 90). In preparing this document, ATSDR's Cooperative Agreement Partner has collected relevant health data, environmental data, and community health concerns from the Environmental Protection Agency (EPA), state and local health and environmental agencies, the community, and potentially responsible parties, where appropriate. This document represents the agency's best efforts, based on currently available information, to fulfill the statutory criteria set out in CERCLA section 104 (i)(6) within a limited time frame. To the extent possible, it presents an assessment of potential risks to human health. Actions authorized by CERCLA section 104 (i)(11), or otherwise authorized by CERCLA, may be undertaken to prevent or mitigate human exposure or risks to human health. In addition, ATSDR's Cooperative Agreement Partner will utilize this document to determine if follow-up health actions are appropriate at this time.

This document has previously been provided to EPA and the affected state in an initial release, as required by CERCLA section 104 (i) (6) (H) for their information and review. Where necessary, it has been revised in response to comments or additional relevant information provided by them to ATSDR's Cooperative Agreement Partner. This revised document has now been released for a 60-day public comment period. Subsequent to the public comment period, ATSDR's Cooperative Agreement Partner will address all public comments and revise or append the document as appropriate. The public health assessment will then be reissued. This will conclude the public health assessment process for this site, unless additional information is obtained by ATSDR's Cooperative Agreement Partner which, in the agency's opinion, indicates a need to revise or append the conclusions previously issued.

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Please address comments regarding this report to:

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1-800-CDC-INFO or  
Visit our Home Page at: <http://www.atsdr.cdc.gov>

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## Foreword

The Florida Department of Health (DOH) evaluates the public health threat of hazardous waste sites through a cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ATSDR) in Atlanta, Georgia. This health consultation is part of an ongoing effort to evaluate health effects associated with the Orlando Gasification Plant hazardous waste site in Orlando, Florida. The DOH evaluates site-related public health issues through the following processes:

- **Evaluating exposure:** DOH scientists review available information about environmental conditions at the site. The first task is to find out how much contamination is present, where it is on the site, and how human exposures might occur. The U.S. Environmental Protection Agency (EPA) provided the information for this assessment.
- **Evaluating health effects:** If DOH finds evidence that exposures to hazardous substances are occurring or might occur, their scientists will determine whether that exposure could be harmful to human health. DOH focuses this report on public health; that is, the health impact on the community as a whole, and bases it on existing scientific information.
- **Developing recommendations:** In this report, the DOH outlines, in plain language, its conclusions regarding any potential health threat posed by contaminated soil, groundwater, and vapor, and offers recommendations for reducing or eliminating human exposure to contaminants. The role of the DOH in dealing with hazardous waste sites is primarily advisory. For that reason, the evaluation report will typically recommend actions for other agencies, including the U.S. EPA and the Florida Department of Environmental Protection (FDEP). If, however, an immediate health threat exists or is imminent, DOH will issue a public health advisory warning people of the danger, and will work to resolve the problem.
- **Soliciting community input:** The evaluation process is interactive. The DOH starts by soliciting and evaluating information from various government agencies, individuals or organizations responsible for cleaning up the site, and those living in communities near the site. DOH shares any conclusions about the site with the groups and organizations providing the information. Once DOH prepares an evaluation report, they seek feedback from the public.

*If you have questions or comments about this report, please write to us at*

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Division of Disease Control and Health Protection  
4052 Bald Cypress Way, Bin # A-12  
Tallahassee, FL 32399-1720

*Or, call us at (850) 245-4401 or toll-free in Florida: 1-877-798-2772*



## Summary

INTRODUCTION	<hr/> <p>At the Orlando Gasification Plant hazardous waste site, the Florida Department of Health (DOH) and the U.S. Agency for Toxic Substances and Disease Registry's (ATSDR) top priority is to ensure nearby residents have the best information to safeguard their health. The purpose of this public health assessment report is to assess the public health threat from toxic chemicals in groundwater, soil, and air at the former Orlando Gasification Plant hazardous waste site. The U.S. Environmental Protection Agency (EPA) requested this assessment.</p> <p>The Orlando Gasification Plant site is north and south of 500-600 West Robinson Street in Orlando, Florida. Between 1887 and 1960, the site was an active manufactured gas plant. The owners used coal to make gas for use in lamps and stoves. They also made a number of by-products. Over time, the plant polluted on-site soil and groundwater. In 1960, after natural gas came to the city, the owners closed and took down the plant. Since then, groundwater pollution has spread to almost a mile northeast of the site.</p>
OVERVIEW	<hr/> <p>DOH reached five conclusions regarding the Orlando Gasification Plant Site.</p>
CONCLUSION #1	<hr/> <p>DOH concludes that incidental ingestion (swallowing) of contaminants in on-site surface soils is not expected to harm workers' health.</p>
BASIS FOR DECISION #1	<hr/> <p>Contaminants in the on-site surface soils are below levels likely to harm health.</p>
CONCLUSION #2	<hr/> <p>DOH concludes that incidental ingestion of contaminants in off-site surface soil along West Robinson Street and Chatham Avenue is not expected to harm the health of passersby.</p>
BASIS FOR DECISION #2	<hr/> <p>Contaminants in the off-site surface soils along West Robinson Street and Chatham Avenue near the site are below levels likely to harm health.</p> <hr/>

CONCLUSION #3	DOH concludes that incidental ingestion of contaminants in surface soil along the railroad track north of the site is not expected to harm trespassers' health.
BASIS FOR DECISION #3	Contaminants in the surface soils along the railroad track north of the site are below levels likely to harm health.
CONCLUSION #4	DOH concludes that since people do not come into contact with contaminants in groundwater or on-site or nearby subsurface soils, they will not harm people's health.
BASIS FOR DECISION #4	Buildings and asphalt cover most of the site. The area near the site is also mostly covered. Therefore, people will not contact subsurface soils. Drinking water in the area is supplied by the Orlando Utilities Commission. The water comes from deep Floridan Aquifer wells 1 mile northeast of the site, so it is not affected. There are no known private drinking water wells within a mile of the site.
CONCLUSION #5	DOH is not able to conclude whether chemicals are present in indoor air that may harm current or future on-site workers because indoor air data is not available.
BASIS FOR DECISION #5	While available on-site soil vapor and radon gas data does not indicate that subslab gas is migrating into onsite buildings at levels of concern, because of the uncertainty in these data, a health conclusion cannot be made in the absence of indoor air data. Evaluating seasonal and spatial variability is also necessary to understand reasonable maximum exposures. Actions taken as part of the Record of Decision (ROD) in Appendix A will help protect against harmful vapor intrusion exposures.
NEXT STEPS	Indoor air sampling is recommended to evaluate exposures in buildings currently onsite. DOH recommends health protective measures and monitoring during cleanup. Vapor mitigation systems may be appropriate when designing and constructing new buildings on site properties. Establish operation, maintenance and monitoring plans for any vapor mitigation systems that may be installed in the future.

DOH will:

- Share this report with nearby residents via a community update to the larger community to summarize its findings and recommendations.
- Solicit public comments on this draft report as well as collect any additional health concerns and address both in the final report.
- Hold an open house to explain the findings to the residents.
- Consider review of new data by request.

#### LIMITATIONS OF FINDINGS

All health risk assessments, to varying degrees, require the use of assumptions, judgments, and incomplete data. These contribute to the uncertainty of the final risk estimates. Some more important sources of uncertainty in this public health assessment include environment sampling and analysis, exposure parameter estimates, use of modeled (average) data, and present toxicological knowledge. We may overestimate or underestimate risk because of these uncertainties. This public health assessment does not represent an absolute estimate of risk to persons exposed to chemicals at or near the Orlando Gasification site.

#### FOR MORE INFORMATION

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If you have concerns about your health or the health of your children, you should contact your health care provider. You may also call the DOH toll-free at 877-798-2772 and ask for information about the former Orlando Gasification Plant hazardous waste site.



## Background and Statement of Issues

The purpose of this public health assessment report is to assess the public health threat from toxic chemicals in groundwater, soil, and air at the former Orlando Gasification Plant hazardous waste site. The U.S. Environmental Protection Agency (EPA) requested this assessment. The former Orlando Gasification Plant site is in downtown Orlando, Florida, on the north and south sides of West Robinson Street (Figure 1).

The former Orlando Gasification Plant site is in a mixed commercial, industrial, and residential area. The site is currently split among several property owners and businesses including: Creative Packaging; Pierce; Clark; the Natasa-Murdinski-Clark Family Trust; Tampa Electric Company; and the City of Orlando (Figure 2). The site is bordered to the north by railroad tracks and the University of Central Florida's downtown campus. The site is bordered to the east by the more commercial properties including a Florida Department of Law Enforcement (FDLE) State Regional Service Center and a plumbing company. The site is bordered to the south by a residential area, including a church, a city park and single family homes. The site is bordered to the west by commercial buildings as well as multi-family and single family homes. There were no reports of other hazardous waste sites that would impact results in the immediate area.

Between 1887 and 1960, the site was an active manufactured gas plant (MGP). Initially the owners limited operations to the area north of West Robinson Street. In 1925, owners expanded the plant to include the area south of West Robinson Street. They brought in coal by the railroad to produce "water gas," which the City of Orlando used in lamps and stoves. As a result of natural gas becoming available in 1959, the owners shut down and dismantled the MGP in 1960. The manufactured gas process created a number of by-products and contaminants often associated with petroleum. Some of the by-products released into the soil eventually migrated to the groundwater. Investigators detected several contaminants above regulatory limits including: metals, polycyclic aromatic hydrocarbons (PAHs) and volatile organic compounds (VOCs) which include benzene, toluene, ethylbenzene, and xylenes [EVIRON 2011].

In the early 1900s, the City of Orlando installed drainage wells to help prevent flooding during heavy rains. The City installed these wells to approximately 250 feet below ground surface (bgs) into the Upper Floridan Aquifer. It is thought that these drainage wells, some of which were in the immediate area of the site, contributed to the presence of site contaminants in the Upper Floridan Aquifer [ARCADIS 2009].

Five Upper Floridan Aquifer non-potable wells (four irrigation wells and one coolant well) were located within a mile of the site. Laboratory analysis detected some contaminants in three of the wells; however, the detection levels were below screening values for drinking water [ARCADIS 2009]. The report did not specify if the contamination found in these wells were site-related.

Area residents and businesses receive their drinking water from the Orlando Utilities Commission. The principle source of drinking water in the area is the Lower Floridan Aquifer, approximately 1,100 to 1,500 feet bgs. The Upper and Lower Floridan Aquifers are separated by an approximately 500 foot thick semi-confining unit, giving the Lower Floridan Aquifer protection from the migration of contaminants [Lichtler, 1968]. The nearest municipal well is located approximately 1 mile northeast of the site. This well is part of the Highland Avenue well field and is installed in the Lower Floridan Aquifer. The nearby public wells are tested quarterly and have not had detections of site-related contaminants. It appears that in the surficial and Upper Floridan zones, site-related groundwater contamination has stabilized or is declining [ARCADIS 2009].

The general direction of surficial groundwater flow in the area of the site is toward the north. In the Upper Florida Aquifer, groundwater flow is to the northeast in this area [ENVIRON 2011].

Soil and groundwater contamination, including light non-aqueous phase liquids (LNAPLs), exists beneath some on-site buildings. As a result, soil vapor intrusion is a potential source of indoor air contamination [ENVIRON 2011].

On July 8, 2013, the U.S. EPA hosted a community meeting for local residents and other interested parties to present the agency's proposed plans for cleanup at the Orlando Gasification Plant site. In September of 2013 EPA released a Record of Decision [EPA 2013c] that details the proposed cleanup plans. The selected remediation plan can be found in Appendix A.

This assessment considers health concerns of nearby workers, residents, pedestrians and trespassers and explores possible associations with site-related contaminants. This assessment requires the use of assumptions, judgments, and incomplete data. These factors contribute to uncertainty in evaluating the health threat. Assumptions and judgments in this assessment err on the side of protecting public health and may overestimate the risk to public health.

## ***Site Description***

The former Orlando Gasification Plant site is a collection of seven parcels totaling approximately 4 acres between the 500 and 600 block of West Robinson Street. The parcels have six different owners. West Robinson Street and the right-of-way are owned by the City of Orlando. The Tampa Electric Company owns two parcels, both north and south of West Robinson Street. These parcels are made up of offices, parking, equipment storage, and a natural gas distribution station. Additional parcels include an FDLE vehicle storage area, a plastics company, and other commercial businesses. All of the smaller properties have limited access and are enclosed by some form of fencing. Asphalt, concrete and buildings cover the vast majority of surface soil at the site.

On April 10, 2013, representatives from the DOH visited the site. The Department noted the right-of-way along West Robinson Street was the only unpaved and unrestricted part of the site. Buildings, pavement, or fences restrict access to the soil on all other parts of the site (Figure 3).

## ***Demographics***

DOH examines demographic and land use data to identify sensitive populations, such as young children, the elderly and women of childbearing age, to determine whether these sensitive populations are exposed to any potential health risks. Demographics also provide details on population mobility and residential history in a particular area. This information helps DOH evaluate how long residents might have been exposed to contaminants.

Approximately 13,415 people live within a 1.0-mile radius of the site. Forty-eight percent (48%) are white, 36% are African-American, 12% are of Hispanic origin, and 4% represent other racial or ethnic groups. Thirteen percent (13%) are less than 18 years old, and 87% are older than 18. Forty two percent (42%) have a high school diploma or less, and 58% have at least some college. Eighty five percent (85%) speak only English, and 62% make less than \$50,000 a year (EPA 2010).

## ***Land Use***

Land use north and east of the former Orlando Gasification Plant site is predominantly commercial and industrial. Land use south of the site is predominantly residential. Land use west of the site is predominantly residential with commercial business along parts of West Robinson Street.

## **Community Health Concerns**

Residents of the neighborhood near the former Orlando Gasification Plant hazardous waste site are concerned about the health risk from contaminated drinking water; the site's potential contribution to asthma; and the health risk from dust, vapors and smells as a result of exposed subsurface soil and groundwater that may arise during future EPA remedial actions.

The Orlando Utilities Commission (OUC) provides drinking water for properties near the site. The closest well field to the site is approximately 1 mile northeast of the facility. The OUC has sampled these wells on a quarterly basis and has not detected site-specific contaminants of concern. In addition, EPA emphasized that all proposed cleanup plans would include procedures to protect public health during any cleanup at a July 8, 2013 public meeting.

## **Discussion**

### ***Environmental Data***

#### **Soil**

Consultants collected on-site surface soils from beneath impervious material, such as asphalt, as well as from exposed soil.

Consultants collected a total of 42 samples from exposed on-site and off-site surface soil [JWC 2002, BBL 2006, and ARCADIS 2009]. See Figures 4 and 5 for sample locations. They analyzed these samples for metals, PAHs, VOCs, and cyanide. Additionally, they analyzed three on-site surface-soil samples (SS-11, SS-12, and SS-14) for organic pesticide compounds. Laboratory analysis found most of the surface soil samples contaminated with arsenic and PAHs above screening guidelines (Table 5).

To evaluate on-site soil, consultants collected 12 on-site surface samples (SS-1 to SS-6, SS-8 and SS-10 to SS-14) from 0 to 6 inches for analysis (Table 5). Figure 4 shows the 12 sample locations. Consultants collected 11 residential surface samples (SS-29 to SS-39) from 0 to 6 inches for analysis of off-site surface soil (Table 7). Figure 4 shows the 11 sample locations. Consultants also collected seven commercial surface samples (SS-7, SS-9, SS-15, SS-16 and SS-40 to SS-42) from 0 to 6 inches for analysis of off-site surface soil along West Robinson Street and Chatham Avenue (Table 10). Figure 4 shows the seven sample locations.

Due to an initial laboratory result of 485 mg/kg of lead for the residential surface soil sample collected at location SS-37 on January 25, 2006, in September 2015 the City of Orlando retested location SS-37. Five surface soil samples were collected with results between 16 mg/kg and 75 mg/kg of lead. For this public health assessment, consultants for the EPA and responsible parties have adequately characterized surface-soil quality.

#### **Soil Vapor**

In January 2006, consultants for the EPA collected one ambient air and 12 soil vapor samples and analyzed for benzene, toluene, ethylbenzene, xylenes, naphthalene, and VOCs. They collected the samples from usually within 10 feet of the outside of the buildings [ARCADIS 2009].

In October 2010, consultants for the responsible party collected eight additional soil gas samples for benzene, toluene, ethylbenzene, xylenes, naphthalene, and VOCs analyses. They collected soil gas samples beneath (2 to 2.5 feet bgs) the slabs of three on-site buildings (A, B, and C) (Figure 6). Building B was demolished in 2016. Building A is an open-air structure and Building C is used for offices [ENVIRON 2011].



To determine the level at which vapors intrude into the buildings, consultants used radon as a tracer gas, although radon may act differently than the gases of concern. Technicians measured radon levels in the sub-slab soil gas and in the indoor air at the same location. Although EPA has determined vapor intrusion is not a likely current human health risk, this assessment considers it a “potential exposure pathway” because building foundations could possibly crack or become damaged during site remedial actions, building modifications, or changing utility lines allowing vapor intrusion.

## ***Pathway Analyses***

Chemical contamination in the environment can harm your health but only if you have contact with those contaminants (exposure). Without contact or exposure, there is no harm to health. If there is contact or exposure, how much of the contaminants you contact (concentration), how often you contact them (frequency), for how long you contact them (duration), and the danger level of the contaminant (toxicity) all determine the risk of harm.

Knowing or estimating the frequency with which people could have contact with hazardous substances is essential to assessing the public health importance of these contaminants. The method for assessing whether a health hazard exists to people is to determine whether there is a completed exposure pathway from a contaminant source to a receptor population and whether exposures to contamination are high enough to be of health concern.

An exposure pathway is a series of steps starting with the release of a contaminant in environmental media and ending at the interface with the human body. A completed exposure pathway consists of five elements:

1. A source of contamination like a hazardous waste site.
2. An environmental medium like air, water or soil that can hold or move the contamination.
3. A point where people come into contact with a contaminated medium like water at the tap or soil in the yard.
4. An exposure route like ingesting (contaminated soil or water) or breathing (contaminated air).
5. A population who could be exposed to contamination like nearby residents.

Generally, the ATSDR/DOH consider three exposure categories: 1) completed exposure pathways; that is, all five elements of a pathway are present; 2) potential exposure pathways; that is, one or more of the elements may not be present, but information is insufficient to eliminate or exclude the element; and 3) eliminated exposure pathways; that is, a receptor population does not come into contact with contaminated media. Exposure pathways are used to evaluate specific ways in which people were, are, or will be exposed to environmental contamination in the past, present, and future.

## **Completed Exposure Pathway**

For this assessment, DOH evaluated the long-term health threat from incidental ingestion (swallowing) of very small amounts of surface soil (0-6 inches deep). For this completed pathway, the former Orlando Gasification Plant hazardous waste site is the source. Activities involved in the manufacture and disposal of water gas and its by-products are thought to have caused contamination of on-site soil. Surface soil is the environmental medium. On-site surface soil is the exposure point. DOH also looked at surface soils: 1) off-site residential, 2) off-site commercial, adjacent to the site and along the railroad tracks and 3) off-site commercial, along West Robinson Street and Chatham Avenue near the site. Incidental ingestion, accidentally swallowing very small amounts of soil, is the exposure route. On-site workers, nearby residents, pedestrians and trespassers are the exposed population (Table 1).

## **Potential Exposure Pathway**

For this assessment, DOH evaluates the long-term health threat from vapor intrusion into the air of on-site buildings. For this potential exposure pathway, the former Orlando Gasification Plant hazardous waste site is the source. Spills and improper disposal of by-products and waste material have contaminated the on-site soil and groundwater. Some of these contaminants may evaporate as vapors (the environmental medium) and possibly travel upward into buildings, making indoor air the possible point of human exposure. Breathing the air inside these buildings would be the exposure route. On-site workers would be the exposed populations (Table 2).

## **Eliminated Exposure Pathways**

Incidental ingestion of sub-surface soil and drinking or showering with water from local private or municipal drinking water wells are eliminated exposure pathways (Table 3).

There is no evidence of exposure to sub-surface soils at or near the site. Cement, asphalt and buildings cover most of on-site sub-surface soil and access is restricted to those areas not covered. There are currently no businesses conducting excavation or other activities that might regularly expose people to subsurface soil on or near the site.

Drinking and showering with water from nearby private or municipal wells are also eliminated exposure pathways. Thirteen municipal wells are located within a 3-mile radius of the site. These municipal wells were installed between 1951 and 1964. There are no known private or municipal drinking water wells within 1 mile of the former Orlando Gasification Plant site. Residents in this area get their drinking water from the municipal supply which is located 1 mile northeast of the site and has been tested at least once per quarter for VOCs since 1993.

## ***Identifying Contaminants of Concern***

DOH compares the maximum concentrations of contaminants found at a site to ATSDR and other comparison values. Comparison values are specific for the medium contaminated (soil, water, air, etc.). DOH screens the environmental data using these comparison values:

- ATSDR Cancer Risk Evaluation Guide (CREG)
- ATSDR Environmental Media Evaluation Guides (EMEGs)
- ATSDR Reference Media Evaluation Guides (RMEGs)
- ATSDR Minimal Risk Level (MRL)
- Florida DEP Soil Cleanup Target Levels (SCTLs)
- EPA Maximum Contaminant Levels (MCLs)
- EPA Lifetime Health Advisory (LTHA)
- EPA Reference Concentration for Chronic Inhalation Exposure (RfC)
- Other guidelines

When determining which comparison value to use, DOH follows ATSDR's general hierarchy and uses professional judgment.

DOH selects for further evaluation contaminants with maximum concentrations above a comparison value. Comparison values, however, are not thresholds of toxicity. DOH and ATSDR do not use them to predict health effects or to establish clean-up levels. A concentration above a comparison value does not necessarily mean harm will occur. It does indicate, however, the need for further evaluation.

Maximum contaminant concentrations below comparison values are not likely to cause illness, and DOH/ATSDR does not evaluate them further.

By comparing the highest measured concentrations in soil and indoor air to ATSDR and EPA screening guidelines, DOH selected arsenic, benzo(a)pyrene toxic equivalents (BaP TEq) as a measurement for PAHs, polychlorinated biphenyls (PCBs) -1260, and lead as contaminants of concern (COCs). DOH will reconsider COCs for indoor air if additional testing is conducted.

Selection of these contaminants does not necessarily mean there is a public health risk. Rather, DOH selected these contaminants for closer scrutiny. Concentrations of other contaminants are below screening guidelines and are not likely to cause illness. DOH/ATSDR does not evaluate these contaminants further.

## **Arsenic**

Arsenic is a naturally-occurring metal widely distributed in soil. Scientists usually find it combined with oxygen, chlorine, and sulfur. Most arsenic compounds have no smell or special taste [ATSDR 2007].

Arsenic, like most metals, is not well absorbed through the skin. If you get arsenic-contaminated soil on your skin, only a small amount will go through your skin into your body, so skin contact is usually not a health risk [ATSDR 2007]. The lack of air monitoring data prevents an evaluation of the risk from breathing arsenic-contaminated dust.

State and federal environmental agencies base their arsenic cleanup standards on workplace studies and laboratory animal studies. Because of uncertainties in these studies, their cleanup standards include large safety factors to ensure public health. Although concentrations slightly above these cleanup standards may not necessarily cause harm, the responsible party should clean up the soil to protect public health.

## **Dibenz(a,h)anthracene (DBA)**

DBA is a polycyclic aromatic hydrocarbon (PAH). No commercial production or use of DBA is known. It occurs as a component of coal tars, shale oils, and soot and has been detected in gasoline engine exhaust, coke oven emissions, cigarette smoke, charcoal broiled meats, vegetation near heavily traveled roads, and surface water and soils near hazardous waste sites [RAIS 1997].

No human studies were available to evaluate the toxicity of DBA. In animals, depressed immune responses were observed in mice following single or multiple injections of DBA. No epidemiologic studies or case reports addressing the carcinogenicity of DBA in humans were available. In animals, DBA has produced tumors by different routes of administration, having both local and systemic carcinogenic effects.

## **Lead**

Lead is a naturally-occurring bluish-gray metal found in small amounts in the soil. Lead can be found in all parts of our environment. Much of it comes from human activities including burning fossil fuels, mining, and manufacturing. Because of health concerns, lead from paints, ceramic products, caulking, and pipe solder has been dramatically reduced in recent years. In 1996 the government banned the use of lead as an additive to gasoline in the United States.

Adults and children may be exposed to lead by hand-to-mouth contact after exposure to lead-containing soil or dust. Most exposure to lead comes from accidental ingestion rather than dermal exposure. Environmental exposure to lead has long been recognized as

a public health problem particularly among children. Excessive concentrations of lead in soil have been shown to increase blood lead levels in young children [ATSDR 2007b].

Lead, like most metals, is not well absorbed through the skin. Soil that contains lead may get on your skin, but only a small portion of the lead will pass through your skin and enter your blood. The only kinds of lead compounds that easily penetrate the skin are the additives in leaded gasoline, which is no longer sold to the general public. Therefore, the general public is not likely to encounter lead that can enter through the skin [ATSDR 2007b]. The lack of air monitoring data prevents an evaluation of the risk from breathing lead-contaminated dust.

Exposure to lead can happen from breathing workplace air or dust, eating contaminated foods, or drinking contaminated water. Children can be exposed from eating lead-based paint chips or playing in contaminated soil. Lead can damage the nervous system, kidneys, and reproductive system. Signs and symptoms associated with lead toxicity include decreased learning capacity and memory, lowered Intelligence Quotient (IQ), speech and hearing impairments, fatigue and lethargy.

Protecting children from exposure to lead is important to lifelong good health. No safe blood lead level in children has been identified. Even low levels of lead in blood have been shown to affect IQ, ability to pay attention, and academic achievement. And effects of lead exposure cannot be corrected. The goal is to prevent lead exposure to children before they are harmed. There are many ways parents can reduce a child's exposure to lead. The most important is stopping children from coming into contact with lead [CDC 2012].

Adults and children who incidentally ingest surface soil could experience noncancer illnesses. DOH used EPA's Integrated Exposure Uptake Biokinetic (IEUBK) model to estimate the possible blood lead levels of children who are exposed daily to surface soil [EPA 2013]. It is important to note that there are uncertainties and limitations in the IEUBK model. One limitation is the inability to decrease the exposure frequency from 365 days per year. Another limitation is that it can only be used to calculate blood lead levels for children.

## **Polycyclic Aromatic Hydrocarbons (PAHs)**

PAHs are a group of over 100 different chemicals that are formed during the incomplete burning of coal, oil and gas, garbage, or other organic substances like tobacco or charbroiled meat. PAHs are usually found as a mixture containing two or more of these compounds, such as soot.

PAHs detected in soils at the site include anthracene, benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(g,h,i)perylene, benzo(a)pyrene, chrysene, dibenzo(a,h)anthracene, fluoranthene, fluorene, indeno(1,2,3-c,d)pyrene, phenanthrene and pyrene. To evaluate toxicity, ATSDR relates the toxicities of the

carcinogenic PAH family members to the toxicity of BaP. They estimate carcinogenic activity relative to BaP as the potency equivalency factor, or PEF [OEHHA 1993]. PEFs are found in Appendix D. To determine the PAH toxicity equivalent (TEQ), concentrations of carcinogenic PAHs other than BaP are multiplied by their respective PEF and then added to the concentration of BaP. ATSDR considers the PAH TEQ concentration the most valid measure of cancer-producing potency of a complex mixture of PAH compounds.

Animal studies have shown that PAHs can cause harmful effects on the skin, body fluids, and ability to fight disease after both short- and long-term exposure. But these effects have not been seen in people. The Department of Health and Human Services (DHHS) has determined that some PAHs may reasonably be expected to be carcinogens [ATSDR 1995]. Because health scientists believe PAHs may cause cancer through a mutagenic mode, ATSDR and DOH use age-dependent adjustment factors to estimate the increased cancer risk.

## **Polychlorinated Biphenyls (PCBs)**

PCBs are a mixture of individual chemicals which are no longer produced in the United States, but are still found in the environment. PCBs have been used as coolants and lubricants in transformers, capacitors, and other electrical equipment because they do not burn easily and are good insulators. PCBs have no known smell or taste. Many commercial PCB mixtures are known in the U.S. by the trade name Aroclor.

Health effects that have been associated with exposure to PCBs include acne-like skin conditions in adults and neurobehavioral and immunological changes in children. The DHHS has concluded that PCBs may reasonably be anticipated to be carcinogens. The EPA and the International Agency for Research on Cancer (IARC) have determined that PCBs are probably carcinogenic to humans [ATSDR 2000].

## ***Public Health Implications***

Health scientists look at what chemicals are present and in what amounts. They compare those amounts to health guidelines. These guidelines are set far below known or suspected levels associated with health effects. DOH uses guidelines developed to protect children. If chemicals are not present at levels high enough to harm children, they would not likely harm adults.

This public health assessment also considers health concerns of nearby residents and explores possible associations with site-related contaminants. This assessment requires the use of assumptions and judgments, and relies on incomplete data. These factors contribute to uncertainty in evaluating the health threat. Assumptions and judgments in the assessment of the site's impact on public health err on the side of protecting public health and may overestimate the risk.

DOH estimates the health risk for individuals exposed to the highest measured level of contamination. DOH provides site-specific public health recommendations on the basis of toxicological literature, levels of environmental contaminants, evaluation of potential exposure pathways, duration of exposure, and characteristics of the exposed population. Whether a person will be harmed depends on the type and amount of contaminant, how they are exposed, how long they are exposed, how much contaminant is absorbed, genetics, and individual lifestyles.

After identifying contaminants of concern, DOH evaluates exposures by estimating daily doses for children and adults. Kamrin [1988] explains the concept of dose as follows:

“...all chemicals, no matter what their characteristics, are toxic in large enough quantities. Thus, the amount of a chemical a person is exposed to is crucial in deciding the extent of toxicity that will occur. In attempting to place an exact number on the amount of a particular compound that is harmful, scientists recognize they must consider the size of an organism. It is unlikely, for example, that the same amount of a particular chemical that will cause toxic effects in a 1-pound rat will also cause toxicity in a 1-ton elephant.

Thus instead of using the amount that is administered or to which an organism is exposed, it is more realistic to use the amount per weight of the organism. Thus, 1 ounce administered to a 1-pound rat is equivalent to 2,000 ounces to a 2,000-pound (1-ton) elephant. In each case, the amount per weight is the same; 1 ounce for each pound of animal.”

This amount per weight is the *dose*. Toxicology uses dose to compare toxicity of different chemicals in different animals. DOH uses the units of milligrams (mg) of contaminant per kilogram (kg) of body weight per day (mg/kg/day) to express doses in this assessment. A milligram is 1/1,000 of a gram (3-4 grains of rice weigh approximately 100 mg); a kilogram is approximately 2 pounds.

To calculate the daily doses of each contaminant, the DOH uses standard factors for dose calculation [ATSDR 2005; EPA 1997]. DOH assumes that people are exposed daily to the maximum concentration measured and makes the health protective assumption that 100% of the ingested chemical is absorbed into the body. The percent actually absorbed into the body is likely less.

Noncarcinogens - For an assessment of the noncancer health risk, DOH and ATSDR use the following formula to estimate a dose:

$$D = (C \times IR \times EF \times CF) / BW$$

D = exposure dose (milligrams per kilogram per day or mg/kg/day)

C = contaminant concentration (milligrams per kilogram or mg/kg)

IR = intake rate of contaminated sediment (milligrams per day or mg/day)

EF = exposure factor (unitless)

CF = conversion factor ( $10^{-6}$  kilograms per milligram or kg/mg)

BW = body weight (kilograms or kg)

$$EF = F \times ED / AT$$

EF = exposure factor (unitless)

F = frequency of exposure (days/year)

ED = exposure duration (years)

AT = averaging time (days) (ED x 365 days/year for noncarcinogens; 70 years x 365 days/year for carcinogens)

ATSDR groups health effects by duration of exposure. Acute exposures are those with duration of 14 days or less; intermediate exposures are those with duration of 15 – 364 days; and chronic exposures are those that occur for 365 days or more (or an equivalent period for animal exposures). ATSDR Toxicological Profiles also provide information on the environmental transport and regulatory status of contaminants.

DOH compares contaminant air concentrations directly to air comparison values and other doses reported in the toxicological literature for inhalation exposures.

Children's doses are generally higher than adults are because their ingestion rates of soil and water, and inhalation of air compared with their low body weights exceed those of adults. Therefore, if children are not at risk, then adults are not either. For non-cancer illnesses, DOH first estimates the health risk by comparing the exposure dose for children to chemical-specific minimal risk levels (MRLs).

MRLs are health guidelines that establish exposure levels many times lower than levels where scientists observed no effects in animals or human studies. ATSDR designed the MRL to protect the most sensitive, vulnerable individuals in a population. The MRL is an exposure level below which non-cancerous harmful effects are unlikely, even after daily exposure over a lifetime. Although ATSDR considers concentrations at or below the relevant comparison value reasonably safe, exceeding a comparison value does not imply adverse health effects are likely. If contaminant doses/concentrations are above comparison values, DOH further analyzes exposure variables (for example, duration and frequency), toxicology of the contaminants, past epidemiology studies, and the weight of evidence for health effects. DOH uses chronic MRLs where possible because exposures are usually longer than a year. If chronic MRLs are not available DOH uses intermediate length MRLs [ATSDR 2005].

DOH and ATSDR use the following equation to estimate increased cancer risk:

$$\text{Risk} = D \times \text{SF}$$

Risk = Cancer risk

D = Age specific non-cancer dose (mg/kg/day)

SF = Slope factor (mg/kg-day)<sup>-1</sup>



If the chemical is known to increase cancer risks due to early life exposure, FDOH and ATSDR use the following equation to estimate increased cancer risk:

$$\text{Risk} = D \times \text{SF} \times \text{ADAF}$$

D = Age specific exposure dose (mg/kg/day)

SF = Slope factor (mg/kg-day)<sup>-1</sup>

ADAF = Age Dependent Adjustment Factor

This is a conservative estimate of the increased cancer risk. The actual increased cancer risk is likely lower. Because of large uncertainties in the way scientists estimate cancer risks, the actual cancer may be as low as zero. Cancer risk is usually estimated for lifetime (78 years) exposure. Studies of animals exposed over their entire lifetime are the basis for calculating cancer slope factors. Usually, researchers know little about the cancer risk in animals from less than lifetime exposures. Therefore, we also use lifetime exposure to estimate the cancer risk in people. If there is no cancer slope (potency) factor, DOH/ATSDR cannot quantify the risk.

For noncancer illnesses, DOH first estimates the health risk for children. Because children are smaller and swallow more soil than adults, their exposure is higher. Therefore, if children are not at risk, then adults are not either.

## ***Non-Cancer & Cancer Health Effects, Health Evaluation***

### **Soil**

This assessment only addresses surface soil sample data from 0 to 6 inches bgs and does not include samples taken from 0 to 24 inches bgs. Considering soil samples 0 to 24 inches bgs may underestimate the true concentration of water-insoluble contaminants deposited on and likely to remain at the ground surface where individuals are most likely to come in contact with them.

Because people are not exposed to soil beneath impervious material, DOH evaluated only those samples from exposed soil in this assessment.

### **On-Site Surface Soil – Worker Exposure**

DOH calculations used a soil intake of 100 mg/day, adult worker (outdoor with low soil contact) weighing 80 kg (approximately 176 lb), exposed 5 times per week with an exposure duration of 25 years.

## **Arsenic**

DOH estimated exposure using a maximum on-site soil concentration for arsenic of 25 mg/kg and a relative bioavailability factor of 60% [EPA 2015b].

### **Noncancer illnesses**

A maintenance worker who incidentally ingests very small amounts of surface soil from the site with the highest arsenic levels is unlikely to develop noncancer illnesses. The maximum worker arsenic dose ( $1.9 \times 10^{-5}$  mg/kg/day) is less than ATSDR's chronic MRL ( $3 \times 10^{-4}$  mg/kg/day) and thus unlikely to cause noncancer illnesses (Table 6).

### **Cancer**

Workers who incidentally ingest surface soil with the highest arsenic levels at the site over a 25-year period are at an “extremely low” increased estimated risk of cancer (Table 6). Multiplying the maximum arsenic dose ( $4.3 \times 10^{-6}$  mg/kg/day) by the EPA cancer slope factor ( $1.5 \text{ mg/kg/day}^{-1}$ ) results in an increased estimated cancer risk of 6.4 in a million ( $0.0000064$  or  $6.4 \times 10^{-6}$ ).

To put this into context, the American Cancer Society estimates that one out of every three Americans (or 333,333 in 1,000,000) will be diagnosed with some form of cancer in their lifetime. Adding the estimated increased cancer risk from exposure to arsenic in the surface soil at the Orlando Gasification Plant hazardous waste site would increase the cancer incidence from 333,333 in 1,000,000 to 333,339 in 1,000,000.

## **DBA**

DOH estimated exposure using a maximum on-site soil concentration for DBA of 1.1 mg/kg and 25-year exposure duration. Calculations also used a soil intake of 100 mg/day and an 80 kg (approximately 176 lb) adult worker exposed 5 times per week.

### **Noncancer illnesses**

DOH estimated exposure using the maximum commercial soil concentration for DBA. DOH compared the maximum concentration against the EPA noncarcinogenic screening levels using a noncancer hazard index of 0.1. A maintenance worker who incidentally ingests very small amounts of surface soil with the highest DBA levels is unlikely to develop noncancer illnesses. Health guidelines were not available for dose comparison.

### **Cancer**

Workers who incidentally ingest (swallow very small amounts of) surface soil with the highest DBA levels at the site over a 25-year period are at an “extremely low” increased estimated risk of cancer (Table 6). Multiplying the maximum DBA cancer dose ( $6.2 \times 10^{-8}$  mg/kg/day) by the EPA cancer slope factor ( $7.3 \text{ mg/kg/day}^{-1}$ ) results in an increased estimated cancer risk of 4.5 in 10,000,000 ( $4.5 \times 10^{-7}$ ).

## **Lead**

DOH estimated exposure using a maximum on-site soil concentration for lead of 80 mg/kg.

### **Noncancer illnesses**

Estimated blood lead levels more accurately predict health effects than traditional dose estimates. Using EPA's IEUBK model, DOH estimates that exposure to the highest concentration of lead in surface soil on the site (80 mg/kg) would result in less than 5 micrograms of lead per deciliter blood ( $\mu\text{g}/\text{dL}$ ) in children (Table 9). Blood lead levels in workers are likely less. In general, adults with blood lead levels less than 5  $\mu\text{g}/\text{dL}$  are not likely to suffer any noncancer illness [ATSDR 2007b]. If children live or play on the site in the future, the highest concentration of lead in the on-site surface soil is not a health hazard.

### **Cancer**

The U.S. DHHS has determined that lead is reasonably anticipated to be a human carcinogen based on limited evidence from studies in humans and sufficient evidence from animal studies. EPA has determined that lead is a probable human carcinogen. The IARC has determined that inorganic lead is probably carcinogenic to humans [ATSDR 2007b].

EPA has not established a cancer slope factor for lead. Therefore, DOH was unable to calculate a lifetime increased cancer risk.

## **PAHs**

DOH estimated exposure using a maximum on-site soil concentration for PAHs as measured as a BaP TEQ of 29.54 mg/kg and 25-year exposure duration. Calculations also used a soil intake of 100 mg/day and an 80 kg (approximately 176 lb) adult worker exposed 5 times per week.

### **Noncancer illnesses**

DOH estimated exposure using the maximum commercial soil concentration for each of the noncarcinogenic PAHs (1-methylnaphthalene, 2-methylnaphthalene, acenaphthene, anthracene, fluoranthene, fluorene, naphthalene, and pyrene). DOH compared the maximum concentration against the EPA noncarcinogenic screening levels using a noncancer hazard index of 0.1. A maintenance worker who incidentally ingests very small amounts of surface soil with the highest noncarcinogenic PAH levels is unlikely to develop noncancer illnesses. DOH did not calculate doses for the noncarcinogenic PAHs since all maximum concentrations were below the EPA noncarcinogenic screening levels.

### **Cancer**

Workers who incidentally ingest (swallow very small amounts of) surface soil with the highest BaP TEQ levels at the site over a 25-year period are at a "low" increased estimated risk of cancer (Table 6). Multiplying the maximum BaP TEQ dose ( $8.5 \times 10^{-6}$

mg/kg/day) by the EPA cancer slope factor ( $7.3 \text{ mg/kg/day}^{-1}$ ) results in an increased estimated cancer risk of 6.2 in 100,000 ( $6.2 \times 10^{-5}$ ).

### **PCBs**

DOH estimated exposure using a maximum on-site soil concentration for PCB-1260 of 0.24 mg/kg and 25-year exposure duration. Calculations also used a soil intake of 100 mg/day and an 80 kg (approximately 176 lb) adult worker exposed 5 times per week.

### **Noncancer illnesses**

A maintenance worker who incidentally ingests very small amounts of surface soil from the site with the highest PCBs levels is unlikely to develop noncancer illnesses. The maximum adult PCBs dose ( $2.1 \times 10^{-7} \text{ mg/kg/day}$ ) is less than ATSDR's chronic MRL ( $2 \times 10^{-2} \text{ mg/kg/day}$ ) and thus unlikely to cause noncancer illnesses (Table 6).

### **Cancer**

Workers who incidentally ingest surface soil with the highest PCBs levels at the Orlando Gasification Plant site over a 25-year period are at an “extremely low” increased estimated risk of cancer (Table 6). Multiplying the maximum PCB-1260 dose ( $2.1 \times 10^{-7} \text{ mg/kg/day}$ ) by the EPA cancer slope factor ( $0.04 \text{ mg/kg/day}^{-1}$ ) results in an increased estimated cancer risk of approximately 3 in a billion ( $0.0000000027$  or  $2.7 \times 10^{-9}$ ).

## **Off-Site Surface Soil – Residential Exposure**

DOH estimated exposure using a soil intake of 100 mg/day and an 80 kg (approximately 176 lbs) body weight for adults. DOH used 33 years, the 95<sup>th</sup> percentile for current residence time, for the cancer risk exposure period. DOH took into account the mutagenic effects of DBA and PAHs for children 6 weeks to 16 years of age when estimating the cancer risks for residential exposure. Exposure risks were calculated using the maximum concentration for each contaminant above the screening level. Maximum exposure risks were low to extremely low ( $10^{-5}$  to  $10^{-6}$ ). Combined cancer risks for residential parcels would also be considered to be low to extremely low.

### **Arsenic**

For non-cancer illness, DOH estimated exposure using a maximum residential surface soil concentration of 13 mg/kg and 1-year exposure duration for children 1 to 2 years old. For cancer risk, DOH estimated an increased cancer risk calculation for the 95<sup>th</sup> percentile for current residence time (33 years) over a lifetime exposure (78 years). DOH used a relative bioavailability factor of 60% of the maximum concentration in the dose and risk calculations for arsenic [EPA 2015].

### **Noncancer illnesses**

A child who incidentally ingests very small amounts of residential surface soil with the highest arsenic levels is unlikely to develop noncancer illnesses. The estimated maximum

arsenic dose for a child 1 to 2 years old ( $1.4 \times 10^{-4}$  mg/kg/day) is less than ATSDR's chronic MRL ( $3 \times 10^{-4}$  mg/kg/day) and thus unlikely to cause noncancer illnesses (Table 8). DOH used the 1-to-2 years old period since this is normally the age range when children ingest the largest amount of soil.

### **Cancer risk**

A person who incidentally ingests very small amounts of residential surface soil with the highest arsenic levels near the site over 33 years is at a “very low” increased estimated risk of cancer (Table 8). Multiplying the maximum arsenic dose for 33 years of exposure ( $4.1 \times 10^{-6}$  mg/kg/day) by the EPA cancer slope factor ( $1.5 \text{ mg/kg/day}^{-1}$ ) results in an estimated increased cancer risk of approximately 3 in million ( $3.1 \times 10^{-6}$ ).

To put this into context, the American Cancer Society estimates that one out of every three Americans (or 333,333 in 1,000,000) will be diagnosed with some form of cancer in their lifetime. Adding the estimated increased cancer risk from exposure to arsenic in the surface soil at the Orlando Gasification Plant hazardous waste site would increase the cancer incidence from 333,333 in 1,000,000 to 333,336 in 1,000,000.

### **DBA**

DOH estimated exposure using a maximum residential off-site soil concentration for DBA of 0.51 mg/kg. DOH used 33 years, the 95<sup>th</sup> percentile for current residence time, for the cancer risk exposure period. DOH took into account the mutagenic effects of DBA for children 6 weeks to 16 years of age when estimating the cancer risks for residential exposure.

### **Noncancer illnesses**

DOH estimated exposure using the maximum commercial soil concentration for DBA. DOH compared the maximum concentration against the EPA noncarcinogenic screening levels using a noncancer hazard index of 0.1. A resident who incidentally ingests very small amounts of surface soil with the highest DBA levels is unlikely to develop noncancer illnesses. DOH did not calculate risk for the noncarcinogenic PAHs since all maximum concentrations were below the EPA noncarcinogenic screening levels.

### **Cancer**

Residents who incidentally ingest (swallow very small amounts of) surface soil with the highest DBA levels at the site over a 33-year period are at a “very low” increased estimated risk of cancer (Table 8). Multiplying the maximum DBA cancer dose ( $1.3 \times 10^{-7}$  mg/kg/day) by the EPA cancer slope factor ( $7.3 \text{ mg/kg/day}^{-1}$ ) results in an increased estimated cancer risk of 1.4 in 100,000 ( $1.4 \times 10^{-5}$ ).

### **Lead**

DOH estimated exposure using a maximum residential surface soil concentration for lead of 485 mg/kg for samples collected in 2006. In September 2015 the City of Orlando resampled the only location (SS-37) that exceeded the FDEP Residential soil cleanup

target level (SCTL) of 400 mg/kg. Five surface samples were collected with results between 16 mg/kg to 75 mg/kg. After the resample the maximum concentration for lead was 162 mg/kg at sample location SS-33 (see Figure 4). This is below the DEP SCTL of 400 mg/kg.

### **Noncancer illnesses**

Estimated blood lead levels more accurately predict health effects than traditional dose estimates. Using EPA's IEUBK model, DOH estimates that exposure to the highest concentration of lead (485 mg/kg) in residential surface soil near the Orlando Gasification Plant site would result in greater than 5 µg/dL for children from 6 months to 4 years of age, the highest result being 6.6 µg/dL for children 1 to 2 (Table 9). Calculations show that at the 485 mg/kg concentration, children 4 years of age and older would have blood lead levels less than 5 µg/dL. The CDC considers a blood lead level of 5 µg/dL or greater to be a level of concern for children. Of the 13 residential soil samples collected (includes 2 duplicate samples), only one sample (SS-37 in Figure 4) resulted in a blood lead level greater than 5 µg/dL for children younger than 4 years of age. The remaining 12 residential soil samples had blood lead level results of 3 µg/dL or less.

Results would be less for samples collected in September 2015 since lead concentrations were between 16 mg/kg and 75 mg/kg.

### **Cancer**

The U.S. DHHS has determined that lead is reasonably anticipated to be a human carcinogen based on limited evidence from studies in humans and sufficient evidence from animal studies. EPA has determined that lead is a probable human carcinogen. The IARC has determined that inorganic lead is probably carcinogenic to humans [ATSDR 2007b].

EPA has not established a cancer slope factor for lead with which to quantify an increased cancer risk. Therefore, DOH was unable to calculate an increased cancer risk for lead exposure.

### **PAHs**

For noncancer illness, DOH estimated exposure using a maximum soil concentration for each of the noncarcinogenic PAHs as screening values. For cancer risk, DOH used the maximum BaP TEQ of 0.69 mg/kg to estimate an increased cancer risk calculation for the 95<sup>th</sup> percentile for current residence time (33 years) over a lifetime exposure (78 years). DOH took into account the mutagenic effects of PAHs for children 6 weeks to 16 years of age when estimating the cancer risks for residential exposure.

### **Noncancer illnesses**

DOH estimated exposure using the maximum commercial soil concentration for each of the noncarcinogenic PAHs (1-methylnaphthalene, 2-methylnaphthalene, acenaphthene, anthracene, fluoranthene, fluorene, naphthalene, and pyrene). DOH compared the maximum concentration against the EPA noncarcinogenic screening levels using a

noncancer hazard index of 0.1. A resident who incidentally ingests very small amounts of surface soil with the highest noncarcinogenic PAH levels over a lifetime is unlikely to develop noncancer illnesses. DOH did not calculate risk for the noncarcinogenic PAHs since all maximum concentrations were below the EPA noncarcinogenic screening levels.

#### **Cancer risk**

Residents who incidentally ingest surface soil with the highest BaP TEQ levels of 0.69 mg/kg at the site over 33 years are at a “very low” increased estimated risk of cancer (Table 8). Multiplying the maximum BaP TEQ dose ( $1.8 \times 10^{-7}$  mg/kg/day) by the EPA cancer slope factor ( $7.3 \text{ mg/kg/day}^{-1}$ ) along with age dependent adjustment factors (ADAFs) results in an increased estimated cancer risk of 2 in 100,000 ( $2.0 \times 10^{-5}$ ).

A child who incidentally ingests very small amounts of residential surface soil with the highest BaP TEQ levels is unlikely to develop cancer-related illnesses. The estimated BaP TEQ dose for a child 1 to 2 years old ( $6.1 \times 10^{-6}$  mg/kg/day) is well below the oral NOAELs for PAHs of 1.3 mg/kg/day. ATSDR has not established an MRL for total PAHs. DOH used the 1-to-2 years old period to estimate soil ingestion since this is normally the period when children ingest the largest amount of soil.

#### **PCBs**

PCBs were not found above the detection levels in residential surface soil near the site. Therefore, PCBs are not considered to be a health risk in off-site residential soil.

### **Off-Site Surface Soil (along West Robinson Street and Chatham Avenue) - Pedestrian Exposure**

DOH calculations used a soil intake of 100 mg/day and an 80 kg (approximately 176 lbs) pedestrian exposed four times per week with an exposure duration of 20 years.

#### **Arsenic**

DOH estimated exposure using a maximum soil concentration for arsenic of 1.2 mg/kg and a relative bioavailability factor of 60% [EPA 2015].

#### **Noncancer illnesses**

A pedestrian who incidentally ingests very small amounts of surface soil along West Robinson Street and Chatham Avenue near the Orlando Gasification Plant site with the highest arsenic levels is unlikely to develop noncancer illnesses. The estimated annual arsenic dose ( $5.2 \times 10^{-7}$  mg/kg/day) for a pedestrian is less than ATSDR’s chronic MRL ( $3 \times 10^{-4}$  mg/kg/day) and thus unlikely to cause noncancer illnesses (Table 11).

#### **Cancer risk**

Pedestrians who incidentally ingest surface soil with the highest arsenic levels from commercial soil along West Robinson Street and Chatham Avenue near the Orlando

Gasification Plant site over a 20-year period are at an “extremely low” increased estimated risk of cancer (Table 11). Multiplying the maximum arsenic dose ( $1.3 \times 10^{-7}$  mg/kg/day) by the EPA cancer slope factor ( $1.5 \text{ mg/kg/day}^{-1}$ ) results in an increased estimated cancer risk of 2 in 10 million ( $0.0000002$  or  $2 \times 10^{-7}$ ).

To put this into context, the American Cancer Society estimates that one out of every three Americans (or 3,333,333 in 10,000,000) will be diagnosed with some form of cancer in their lifetime. Adding the estimated increased cancer risk from exposure to arsenic in the surface soil at the Orlando Gasification Plant hazardous waste site would increase the cancer incidence from 3,333,333 in 10,000,000 to approximately 3,333,335 in 10,000,000.

### **DBA**

DOH estimated exposure using a maximum commercial soil concentration for DBA of 0.37 mg/kg and 20-year exposure duration. Calculations also used a soil intake of 100 mg/day and an 80 kg (approximately 176 lbs) pedestrian exposed four times per week.

#### **Noncancer illnesses**

DOH compared the maximum concentration against the EPA noncarcinogenic screening levels using a noncancer hazard index of 0.1. A pedestrian who incidentally ingests very small amounts of surface soil along West Robinson and Chatham Avenue near the site with the highest noncarcinogenic DBA levels is unlikely to develop noncancer illnesses. DOH did not calculate risk for the noncarcinogenic PAHs since all maximum concentrations were below the EPA noncarcinogenic screening levels.

#### **Cancer risk**

Pedestrians who incidentally ingest commercial surface soil with the highest DBA levels near the site over a 20-year period are at an “extremely low” increased estimated risk of cancer (Table 11). Multiplying the maximum DBA dose ( $6.8 \times 10^{-8}$  mg/kg/day) by the EPA cancer slope factor ( $7.3 \text{ mg/kg/day}^{-1}$ ) results in an increased estimated cancer risk of 4.9 in 10,000,000 ( $4.9 \times 10^{-7}$ ).

### **Lead**

DOH estimated exposure using a maximum commercial soil concentration for lead of 110 mg/kg.

#### **Noncancer illnesses**

Estimated blood lead levels more accurately predict health effects than traditional dose estimates. Using EPA’s IEUBK model, DOH estimates that pedestrian exposure to the highest concentration of lead in commercial soil along West Robinson Street and Chatham Avenue near the site (110 mg/kg) would result in a blood lead level of less than 5 µg/dL (Table 9). In general, adults with blood lead levels less than 5 µg/dL are not



likely to suffer any noncancer illness [ATSDR 2007b]. For adult workers, the U.S. OSHA recommends an evaluation when blood lead levels exceed 40 µg/dL.

If children play on these areas, the highest concentration of lead in the commercial surface soil along West Robinson Street and Chatham Avenue near the site should not be considered a health hazard.

### **Cancer risk**

The U.S. DHHS has determined that lead is reasonably anticipated to be a human carcinogen based on limited evidence from studies in humans and sufficient evidence from animal studies. EPA has determined that lead is a probable human carcinogen. The IARC has determined that inorganic lead is probably carcinogenic to humans [ATSDR 2007b].

EPA has not established a cancer slope factor for lead with which to quantify an increased cancer risk. Therefore, DOH was unable to calculate a lifetime excess cancer risk for lead exposure.

### **PAHs**

DOH estimated exposure using a maximum commercial soil concentration for PAHs as measured as a BaP TEQ of 2.5 mg/kg and 20-year exposure duration. Calculations also used a soil intake of 100 mg/day and an 80 kg (approximately 176 lbs) pedestrian exposed four times per week.

### **Noncancer illnesses**

DOH estimated exposure using the maximum commercial soil concentration for each of the noncarcinogenic PAHs (1-methylnaphthalene, 2-methylnaphthalene, acenaphthene, anthracene, fluoranthene, fluorene, naphthalene, and pyrene). DOH compared the maximum concentration against the EPA noncarcinogenic screening levels using a noncancer hazard index of 0.1. A pedestrian who incidentally ingests very small amounts of surface soil along West Robinson and Chatham Avenue near the site with the highest noncarcinogenic PAH levels is unlikely to develop noncancer illnesses. DOH did not calculate risk for the noncarcinogenic PAHs since all maximum concentrations were below the EPA noncarcinogenic screening levels.

### **Cancer risk**

Pedestrians who incidentally ingest commercial surface soil with the highest BaP TEQ levels near the site over a 20-year period are at an “extremely low” increased estimated risk of cancer (Table 11). Multiplying the maximum BaP TEQ dose ( $4.6 \times 10^{-7}$  mg/kg/day) by the EPA cancer slope factor ( $7.3 \text{ mg/kg/day}^{-1}$ ) results in an increased estimated cancer risk of 3.4 in 1,000,000 ( $3.4 \times 10^{-6}$ ).

## **PCBs**

PCBs were not found above the detection levels in commercial soil along West Robinson Street and Chatham Avenue near the Orlando Gasification Plant site. Therefore, PCBs are not considered to be a health risk from these areas.

## **Off-Site Commercial Surface Soil (Along the Railroad Track Northeast and Northwest of the Site) – Trespasser Exposure**

Consultants collected six surface samples (SS-17 to SS-22) from areas along the railroad tracks north of the site at 0 to 6 inches for analysis (Table 12). Figure 5 shows the six sample locations. These areas are almost completely fenced to the north and south of the railroad tracks and are posted with “No Trespassing” signs. DOH did not calculate a cancer risk for this scenario due to the duration and infrequency of the trespass. Noncancer illness calculations used a soil intake of 100 mg/day and a 45 kg (approximately 100 lbs) area trespasser exposed four times per week with an exposure duration of 10 years.

### **Arsenic**

DOH estimated exposure using a maximum soil concentration for arsenic of 39 mg/kg and a relative bioavailability factor of 60% [EPA 2015].

### **Noncancer illnesses**

A trespasser who incidentally ingests very small amounts of surface soil from along the railroad track north of the site with the highest arsenic levels is unlikely to develop noncancer illnesses. The estimated annual arsenic dose ( $3.0 \times 10^{-5}$  mg/kg/day) for a trespasser is less than ATSDR’s chronic MRL ( $3 \times 10^{-4}$  mg/kg/day) and thus unlikely to cause noncancer illnesses (Table 13).

### **Cancer**

A trespasser who incidentally ingest surface soil with the highest arsenic levels from along the railroad track over a 10-year period are at an “extremely low” increased estimated risk of cancer. Multiplying the maximum arsenic dose ( $1.1 \times 10^{-6}$  mg/kg/day) by the EPA cancer slope factor ( $1.5 \text{ mg/kg/day}^{-1}$ ) results in an increased estimated cancer risk of 8.1 in a million ( $0.0000081$  or  $8.1 \times 10^{-6}$ ).

### **DBA**

DOH estimated exposure using the maximum commercial soil concentration for DBA. DOH compared the maximum concentration against the EPA noncarcinogenic screening levels using a noncancer hazard index of 0.1. Calculations also used a soil intake of 100 mg/day and a 45 kg (approximately 100 lbs) area trespasser exposed 4 times per week.

**Noncancer illnesses**

A trespasser who incidentally ingests very small amounts of surface soil with the highest noncarcinogenic PAH levels is unlikely to develop noncancer illnesses. DOH did not calculate risk for the noncarcinogenic PAHs since all maximum concentrations were below the EPA noncarcinogenic screening levels.

**Cancer**

A trespasser who incidentally ingest (swallow very small amounts of) surface soil with the highest DBA levels at the site over a 10-year period are at an “extremely low” increased estimated risk of cancer. Multiplying the maximum DBA cancer dose ( $1.1 \times 10^{-6}$  mg/kg/day) by the EPA cancer slope factor ( $7.3 \text{ mg/kg/day}^{-1}$ ) results in an increased estimated cancer risk of 8.1 in 1,000,000 ( $8.1 \times 10^{-6}$ ).

**Lead**

DOH estimated exposure using a maximum commercial soil concentration for lead of 1200 mg/kg.

**Noncancer illnesses**

Estimated blood lead levels more accurately predict health effects than traditional dose estimates. Using EPA’s IEUBK model, DOH estimates that exposure to the highest concentration of lead in soil along the railroad track north of the site (1200 mg/kg) would result in less than 5 µg/dL in trespassers (Table 9). In general, adults with blood lead levels less than 5 µg/dL are not likely to suffer any noncancer illness [ATSDR 2007b]. For adult workers, the U.S. OSHA recommends an evaluation when blood lead levels exceed 40 µg/dL.

If children trespass on these areas four times a week or less, DOH does not consider the highest concentration of lead in the soil along the railroad track north of the site a health hazard.

**Cancer risk**

The U.S. DHHS has determined that lead is reasonably anticipated to be a human carcinogen based on limited evidence from studies in humans and sufficient evidence from animal studies. EPA has determined that lead is a probable human carcinogen. The IARC has determined that inorganic lead is probably carcinogenic to humans [ATSDR 2007b].

EPA has not established a cancer slope factor for lead with which to quantify an increased cancer risk. Therefore, DOH was unable to calculate a lifetime excess cancer risk for lead exposure.

**PAHs**

DOH estimated exposure using the maximum commercial soil concentration for each of the noncarcinogenic PAHs (1-methylnaphthalene, 2-methylnaphthalene, acenaphthene,

anthracene, fluoranthene, fluorene, naphthalene, and pyrene). DOH compared the maximum concentration against the EPA noncarcinogenic screening levels using a noncancer hazard index of 0.1. Calculations also used a soil intake of 100 mg/day and a 45 kg (approximately 100 lbs) area trespasser exposed 4 times per week.

### **Noncancer illnesses**

A trespasser who incidentally ingests very small amounts of surface soil with the highest noncarcinogenic PAH levels is unlikely to develop noncancer illnesses. DOH did not calculate doses for the noncarcinogenic PAHs since all maximum concentrations were below the EPA noncarcinogenic screening levels.

### **PCBs**

Laboratory analysis did not find PCBs above the detection levels in commercial surface soil along the railroad track north of the Orlando Gasification Plant site. Therefore, DOH does not consider PCBs to be a health risk from these areas.

## **On-Site Vapor Intrusion – Worker Exposure**

Data analysis from January and October soil gas samples showed benzene, 1,3-butadiene, chloroform, and naphthalene above cancer-based soil gas screening levels. Chemicals above screening levels warrant further analysis. The soil gas concentrations were all less than 100 times the soil gas screening levels based on one in a million extra cancer risk.

Radon testing showed very low levels in the indoor air of Buildings A, B, and C (0.24 to 0.92 pCi/L) despite relatively high subslab gas concentrations (692 to 1880 pCi/L). Therefore, the radon data do not support the presence of significant pathways from soil gas to indoor air at the site buildings<sup>1</sup>. However, other pathways, such as sewer gas, generally cannot be ruled out without indoor air sampling.

The lines of evidence collected did not find that indoor air of current on-site buildings are impacted by subsurface contamination. However, data gaps make it impossible to determine for certain. Summertime conditions and variability of soil gas under different regions the buildings are also unknown. Ongoing remediation and taking appropriate precautions during redevelopment will decrease the potential for health hazards. Exposures prior to data collection cannot be evaluated due to lack of past monitoring data.

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<sup>1</sup> Naturally occurring radon may serve as a tracer to help identify those buildings that are more susceptible to soil gas entry than others. The radon concentration in a building is not generally expected to be a good quantitative indicator of indoor air exposure concentrations of vapor-forming chemicals arising from subsurface contamination. Hence, radon measurement is not generally recommended as a proxy for directly measuring vapor-forming chemicals in indoor air. ATSDR recommends concurrently collecting indoor air, sub-slab gas, and outdoor air to evaluate the full vapor intrusion pathway.

The site remedial design is to remove, stabilize, and control migration of the contaminants onsite. Long-term groundwater monitoring is planned. DOH and ATSDR are available to review site safety and monitoring plans for remediation and redevelopment activities upon request.

## **Health Outcome Data**

DOH epidemiologists did not evaluate area cancer rates for two reasons. First, the maximum estimated increased cancer risks for exposure to contaminants of concern in the surface soil on or near this site is “low” to “extremely low.” Second, in addition to the low to extremely low risk, exposure to surface soil is limited due to the restricted access to the site as a result of the site being fenced and mostly covered by structures, asphalt or cement.

## **Child Health Considerations**

In communities faced with air, water, or soil contamination, the many physical differences between children and adults demand special attention. Children could be at greater risk than adults from certain kinds of exposure to hazardous substances. Children play outdoors and sometime engage in hand-to-mouth behaviors that increase their exposure potential. Children are shorter than adults; this means they breathe dust, soil and vapors close to the ground. A child’s lower body weight and higher intake rate results in a greater dose of hazardous substance per unit of body weight. If toxic exposure levels are high enough during critical growth stages, the developing body system of children can sustain permanent damage. Finally, children are dependent on adults for access to housing, for access to medical care, and for risk identification. Thus, adults need as much information as possible to make informed decisions regarding their children’s health.

This assessment takes into account the special vulnerabilities of children. It specifically assesses the health risk for children playing in the surface soil of residential properties near the former Orlando Gasification Plant hazardous waste site. DOH found that children less than 4 years of age exposed to lead via incidental ingestion of surface soil at one sample location are more likely to encounter a level above the level of concern than adults.

## **Conclusions**

DOH reached five conclusions regarding the Orlando Gasification Plant Site. DOH concludes:

1. Incidental ingestion of (swallowing) contaminants in on-site surface soils is not expected to harm workers’ health.

2. Incidental ingestion of contaminants in off-site surface soil along West Robinson Street and Chatham Avenue is not expected to harm the health of passersby.
3. Incidental ingestion of contaminants in surface soil along the railroad track north of the site is not expected to harm trespassers' health.
4. Since people do not come into contact with contaminants in groundwater or on-site or nearby subsurface soils, they will not harm people's health.
5. DOH is not able to conclude whether chemicals are present in indoor air that may harm current or future on-site workers because indoor air data is not available.

## **Recommendations**

Indoor air sampling is recommended to evaluate exposures in buildings currently onsite. Health protective measures and monitoring during cleanup are recommended. Vapor mitigation systems may be appropriate when designing and constructing new buildings on site properties. Establish operation, maintenance and monitoring plans for any vapor mitigation systems that may be installed in the future.

## **Public Health Action Plan**

### ***Actions Planned***

DOH will:

- Share this report with nearby residents via a community update to the larger community to summarize its findings and recommendations.
- Solicit public comments on this draft report as well as collect any additional health concerns and address both in the final report.
- Hold an open house to explain the findings to the residents and extend the public comment period accordingly if response to the update is substantial.
- Consider review of new data by request.

## **Report Preparation**

This Public Health Assessment for the former Orlando Gasification Plant site was prepared by the DOH under a cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with the approved agency methods, policies, procedures existing at the date of publication. Editorial review was completed by the cooperative agreement partner. ATSDR has reviewed this document and concurs with its findings based on the information presented.

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## Appendices



## **Appendix A**

### **Selected Information from EPA Record of Decision (ROD)**

#### **Summary of Remedial Alternative Selection**

#### **Orlando Former Gasification Plant**

**September 2013**





## **Description of Selected Remedy**

The primary components of the Selected Remedy include:

- Excavation and off-site disposal of surface soil containing Site-specific contaminants of concern at concentrations above the cleanup levels
- Excavation and off-site disposal of unsaturated soils in the area(s) along the northern and eastern boundaries of parcel 2 where mobile light non-aqueous phase liquid (LNAPL) was observed during the remedial investigation (RI)
- Implementation of in-situ stabilization and solidification (ISS) in the saturated zone where saturated and/or mobile dense non aqueous phase liquid (DNAPL) was observed in the area(s) along the northern m boundary of the parcel 3
- Removal and off-site disposal of former manufactured gas plant (MGP) structures such as the tar well and associated piping encountered during excavation activities
- Demolition of the building on parcel 2 and off-site disposal of building materials
- Installation of an engineered capping system over the footprint of the "source area" to minimize infiltration
- Backfilling of the excavated areas with clean backfill material and restoration of construction-impacted hardscape areas as needed
- Installation of an ISS wall along the perimeter of the highly impacted soil and groundwater areas also identified as "source area" (where residual MGP impacts were observed) and where groundwater concentrations are greater than or equal to FDEP natural attenuation default concentrations (NADCs)
- Implementation of a short-term groundwater monitoring program subsequent to the construction of the "source area" containment system to determine the extent of the areas within the dissolved groundwater plume where in-situ enhanced bioremediation (ISEB)/biosparging or MNA will be implemented. It is anticipated this groundwater monitoring program will be conducted over a period of approximately three years.
- Implementation of a long-term groundwater monitoring program to assess the effectiveness of the remedy.
- Implementation of institutional controls (ICs) such as restrictive covenants or land and groundwater use restrictions to ensure the effectiveness of the remedy.

Hydraulic control within the containment wall will be achieved by installing subsurface drain(s) inside the ISS wall. The hydraulic control system will be designed to allow for treatment of the contaminated groundwater that flow through the ISS wall via aeration,

activated carbon or other technologies as determined by the EPA. Sampling and monitoring of the treated groundwater will be required to ensure the effectiveness of the treatment technology. The total estimated cost of the Selected Remedy is \$18 million.

### **9.1.1 Institutional Controls (ICs)**

The NCP states that institutional controls, while not actively cleaning up the contamination at the Site, can control exposure, and therefore, are considered to be limited action alternatives. The NCP preamble states: "Institutional controls will usually be used as supplementary protective measures during implementation of groundwater remedies."

Consistent with the RAOs developed for the Site, the specific performance objectives for the institutional controls (ICs) to be implemented at the Site are to prevent human exposure to soil, groundwater, and indoor air with contaminants above levels that pose unacceptable risk and do not allow for unrestricted exposure. EPA will use ICs also to maintain the integrity of the any existing or future monitoring or remediation system. The Potentially responsible parties (PRPs) will be responsible to maintain and monitor the ICs. The FDEP will primarily take responsibility to enforce the ICs according to the ROD. During the remedial design (RD), a Site Management Plan will be developed. This plan will provide the requirements to manage remaining soils that exceed RAOs after remedial construction has been completed. The plan will also provide the implementation and enforcement mechanisms of the identified ICs.

The following generally describes those ICs to be considered for implementation at the Site to achieve the performance objectives:

- Restrictive covenants will be executed by on-site property owners prohibiting the reuse of the properties for residential purposes unless prior written approval is obtained from EPA and FDEP.
- Installation of groundwater extraction wells and the use of groundwater within the contaminated groundwater plume will be prohibited (except for groundwater monitoring wells approved by EPA/FDEP).

### **9.9 Alternative 8 - Soil Excavation with Parcel 2 Building Removal, ISS Containment Wall, Engineered Cap, NAPL Stabilization and/or Excavation and Off-site Disposal, Biosparging (In-Situ enhanced bioremediation), MNA and ICs**

Estimated Capital Cost: \$19.5 million

Estimated O&M Cost: \$1.6 million

Estimated Present Worth Cost: \$18 million

Estimated Time to Achieve RAOs and Cleanup Levels: 20 Years (outside ISS containment wall); > 30 Years (inside ISS containment wall)

Under Alternative 8, biosparging (in-situ enhanced bioremediation) and MNA would be implemented to address the dissolved groundwater plume. Subsequent to the implementation of the "source area" ISS containment wall, a short-term groundwater monitoring program would be developed and implemented. The results of this groundwater monitoring program would be used to determine the extent of the areas where biosparging or MNA would be implemented.

Based on the locations of historical MGP features at the facility and the presence of NAPL observed during the RI activities directly adjacent to the building on parcel 3, it is suspected that substantial saturated coal tar DNAPL may be present beneath the building. As a result, during the design phase, additional investigation would be conducted. If substantial DNAPL is observed during these investigations, the building would be demolished and the NAPL would be addressed as discussed above (excavation and/or ISS).

As discussed in Section 9.1, Site Management Plan, long-term groundwater monitoring, and ICs, would be implemented. In addition, Five-Year review would be conducted to ensure the effectiveness of the remedy. A conceptual layout of Alternative 8 is presented on Figure 14.







## **Appendix B**

### **Tables**





**Table 1. Completed Human Exposure Pathway at the Orlando Gasification Plant Site**

Completed Pathway Name	Completed Exposure Pathway Elements					Time
	Source	Environmental Media	Point of Exposure	Route of Exposure	Exposed Population	
Incidental ingestion (swallowing) of on-site soil	Orlando Gasification Plant	Exposed surface soil	On-site	Incidental ingestion	On-site workers	Past, present, and future
Incidental ingestion of off-site residential soil	Orlando Gasification Plant	Exposed surface soil	Off-site residential areas	Incidental ingestion	Nearby residents	Past, present, and future
Incidental ingestion of off-site commercial soil	Orlando Gasification Plant	Exposed surface soil	Off-site commercial areas south of site	Incidental ingestion	Pedestrians	Past, present, and future
Incidental ingestion of off-site commercial soil	Orlando Gasification Plant	Exposed surface soil	Off-site commercial areas north of the site along the RR tracks	Incidental ingestion	Trespassers	Past, present, and future

**Table 2. Potential Human Exposure Pathways at the Orlando Gasification Plant Site**

Completed Pathway Name	Completed Exposure Pathway Elements					Time
	Source	Environmental Media	Point of Exposure	Route of Exposure	Exposed Population	
Vapor intrusion into air of on-site buildings	Orlando Gasification Plant	Indoor air	Indoor air of on-site buildings	Inhalation	On-site workers	Future

**Table 3. Eliminated Human Exposure Pathways at the Orlando Gasification Plant Site**

<b>Completed Pathway Name</b>	<b>Completed Exposure Pathway Elements</b>				
	<b>Source</b>	<b>Environmental Media</b>	<b>Point of Exposure</b>	<b>Route of Exposure</b>	<b>Exposed Population</b>
On-site subsurface soil	Orlando Gasification Plant	Soil	None	Ingestion	None
Off-site subsurface soil	Orlando Gasification Plant	Soil	None	Ingestion	None
Drinking water from municipal water wells	Orlando Gasification Plant	Deep Groundwater	None	Ingestion	None
Showering with water from municipal water wells	Orlando Gasification Plant	Deep Groundwater	None	Inhalation	None

**Table 4a. Building A<sup>(1)</sup> - Sub-slab Soil Gas Concentrations Above Screening Levels (adjusted 0.03 attenuation value) at the Orlando Gasification Plant Site - October, 2010**

Contaminants	Sub-slab Soil Gas Concentration (µg/m <sup>3</sup> )	Air Screening Level* (unadjusted) (µg/m <sup>3</sup> )	Source of Air Screening Guideline	Air Screening Level* (adjusted) (µg/m <sup>3</sup> )	# Above Screening Level*/Total #
1,2,4-Trimethylbenzene	36	7.3	EPA RSL	2.43E+02	0/1
1,3,5-Trimethylbenzene	16.5	7.3	EPA RSL	2.43E+02	0/1
2-Butanone (Methyl Ethyl Ketone)	11.5	5,000	Rfc	1.67E+05	0/1
2-Propanol	220	730	EPA RSL	2.43E+04	0/1
4-Ethyltoluene	20.5	na		-	-
Acetone	117	31,000	cEMEG	1.03E+06	0/1
Benzene	70.5	0.13	CREG	4.33E+00	<b>1/1</b>
Bromodichloromethane	7.45	na		-	-
Carbon Disulfide	12	930	cEMEG	3.10E+04	0/1
Chloroform	32	0.043	CREG	1.43E+00	<b>1/1</b>
Ethanol	12	na		-	-
Ethyl Benzene	13.5	260	cEMEG	8.67E+03	0/1
Methylene Chloride	8.4	100	CREG	3.33E+03	0/1
Naphthalene	225	0.083	EPA RSL	2.77E+00	<b>1/1</b>
Styrene	13	850	cEMEG	2.83E+04	0/1
Tetrachloroethene	14	3.8	CREG	1.27E+02	0/1
Toluene	82	300	cEMEG	1.00E+04	0/1
Xylenes (total)	80	220	cEMEG	7.33E+03	0/1

Source of data: (ENVIRON 2011)

<sup>(1)</sup> = Building A (Figure 6)

**CREG** = ATSDR cancer risk evaluation guide

**µg/m<sup>3</sup>** = micrograms per cubic meter

\* Screening guidelines only used to select chemicals for further scrutiny, not to the judge the risk of illness.

**na** = not available

**cEMEG** = ATSDR chronic environmental media evaluation guide

**EPA RSL** = Environmental Protection Agency Regional Screening Level (Residential Air)

**Table 4b. Building B<sup>(1)</sup> - Sub-slab Soil Gas Concentrations Above Screening Levels (adjusted 0.03 attenuation value) at the Orlando Gasification Plant Site- October, 2010**

Contaminants	Sub-slab Soil Gas Concentration (µg/m <sup>3</sup> )	Air Screening Level* (unadjusted) (µg/m <sup>3</sup> )	Source of Air Screening Guideline	Air Screening Level* (adjusted) (µg/m <sup>3</sup> )	# Above Screening Level*/Total #
1,2,4-Trimethylbenzene	5.5	7.3	EPA RSL	2.43E+02	0/1
1,3-Butadiene	41	0.033	CREG	1.10E+00	<b>1/1</b>
2,2,4-Trimethylpentane	4.6	na	-	-	-
2-Butanone (Methyl Ethyl Ketone)	34	5,000	Rfc	1.67E+05	0/1
2-Propanol	14	730	EPA RSL	2.43E+04	0/1
4-Ethyltoluene	6.9	na	-	-	-
Acetone	89	31,000	cEMEG	1.03E+06	0/1
Benzene	33	0.13	CREG	4.33E+00	<b>1/1</b>
Carbon Disulfide	69	930	cEMEG	3.10E+04	0/1
Chloroform	22	0.043	CREG	1.43E+00	<b>1/1</b>
Cyclohexane	16	6000	Rfc	2.00E+05	0/1
Ethanol	60	na	-	-	-
Ethyl Benzene	13	260	cEMEG	8.67E+03	0/1
Freon 11	4.6	na	-	-	-
Heptane	25	na	-	-	-
Hexane	48	2,000	cEMEG	6.67E+04	0/1
Methylene Chloride	3.6	100	CREG	3.33E+03	0/1
Styrene	17	850	cEMEG	2.83E+04	0/1
Toluene	42	300	cEMEG	1.00E+04	0/1
Xylenes (total)	25	220	cEMEG	7.33E+03	0/1

Source of data: (ENVIRON 2011)

<sup>(1)</sup> = Building B (Figure 6)

**CREG** = ATSDR cancer risk evaluation guide

**µg/m<sup>3</sup>** = micrograms per cubic meter

\* Screening guidelines only used to select chemicals for further scrutiny, not to the judge the risk of illness.

**na** = not available

**cEMEG** = ATSDR chronic environmental media evaluation guide

**EPA RSL** = Environmental Protection Agency Regional Screening Level (Residential Air)

**Table 4b. Building C<sup>(1)</sup> - Sub-slab Soil Gas Concentrations Above Screening Levels (adjusted 0.03 attenuation value) at the Orlando Gasification Plant Site - October, 2010**

Contaminants	Sub-slab Soil Gas Concentration (µg/m <sup>3</sup> )	Air Screening Level* (unadjusted) (µg/m <sup>3</sup> )	Source of Air Screening Guideline	Air Screening Level* (adjusted) (µg/m <sup>3</sup> )	# Above Screening Level*/Total #
2-Butanone (Methyl Ethyl Ketone)	4.3	5,000	Rfc	1.67E+05	0/1
Acetone	22	31,000	cEMEG	1.03E+06	0/1
Carbon Disulfide	2.9	930	cEMEG	3.10E+04	0/1
Chloroform	21	0.043	CREG	1.43E+00	<b>1/1</b>
Ethyl Benzene	4.2	260	cEMEG	8.67E+03	0/1
Styrene	8.2	850	cEMEG	2.83E+04	0/1
Toluene	3	300	cEMEG	1.00E+04	0/1
Xylenes (total)	21.6	220	cEMEG	7.33E+03	0/1

Source of data: (ENVIRON 2011)

<sup>(1)</sup> = Building C (Figure 6)

**CREG** = ATSDR cancer risk evaluation guide

**µg/m<sup>3</sup>** = micrograms per cubic meter

\* Screening guidelines only used to select chemicals for further scrutiny, not to judge the risk of illness.

**na** = not available

**cEMEG** = ATSDR chronic environmental media evaluation guide

**EPA RSL** = Environmental Protection Agency Regional Screening Level (Residential Air)

**Table 5. Contaminant Concentrations in On-Site Surface Soil (0 to 6 Inches Deep) at the Orlando Gasification Plant Site – April, 2004**

<b>Contaminants</b>	<b>Concentration Range (mg/kg)</b>	<b>Maximum Concentration in Surface Soil (mg/kg) (sample#)</b>	<b>Soil Screening Guideline (mg/kg)*</b>	<b>Source of Screening Guideline</b>	<b># of samples above screening guideline/total # samples</b>
Arsenic	0.5 U - 25	25 (SS-3)	15	RMEG	<b>2/12</b>
DBA	0.02 – 1.1	1.1 (SS-2)	NA	NA	NA
Lead	16 J - 80 J	80J (SS-11)	400*	FDEP Residential SCTL	0/12
PAHs as BaP TEq	0.07 – 29.54	29.54 (SS-2)	0.1	CREG	<b>11/12</b>
PCB	0.1 U - 0.24	0.24 (SS-14)	0.35	CREG	0/12

source of data [ARCADIS 2009]

**BaP TEq** = Benzo(a)Pyrene Toxicity Equivalents

**CREG** = ATSDR cancer risk evaluation guide

**DBA** = Dibenz(a,h)anthracene

**EPA** = US Environmental Protection Agency

**FDEP** = Florida Department of Environmental Protection

**J** = estimated value

**NA** = non-applicable

**PAH** = polycyclic aromatic hydrocarbon

**PCB** = polychlorinated biphenyl

**RMEG** = reference dose media evaluation guide

**SCTL** = soil cleanup target level

**U** = analyte not detected; reporting limit shown

\* Screening guidelines only used to select chemicals for further scrutiny, not to the judge the risk of illness. As no level of lead exposure is safe, ATSDR does not have a soil screening guideline for lead.

**Table 6. Estimated Worker Dose and Increased Cancer Risk From Inadvertent Ingestion of Surface Soil on the Orlando Gasification Plant Site**

Contaminants	Maximum On-Site Soil Concentration (0-6" deep) (mg/kg)	Estimated Worker Maximum Inadvertent Soil Ingestion Noncancer Dose (mg/kg/day)	ATSDR Minimal Risk Level (mg/kg/day)	Estimated Worker Maximum Inadvertent Soil Ingestion Cancer Dose (mg/kg/day)	Oral Cancer Slope Factor (mg/kg-day) <sup>-1</sup>	Source of Oral Cancer Slope Factor	Estimated Increased Cancer Risk
Arsenic	25 <sup>(1)</sup>	$1.9 \times 10^{-5}$	$3 \times 10^{-4}$ (chronic)	$4.3 \times 10^{-6}$	1.5	EPA IRIS	$6.4 \times 10^{-6}$ (extremely low)
DBA	1.1	$1.9 \times 10^{-7}$	none ***	$6.2 \times 10^{-8}$	4.1	Cal EPA	$4.5 \times 10^{-7}$ (extremely low)
Lead	80 J		none **	NA	none	NA	NA
PAHs as BaP TEq	29.54	$2.6 \times 10^{-5}$	none ***	$8.5 \times 10^{-6}$	7.3	EPA IRIS	$6.2 \times 10^{-5}$ (very low)
PCBs	0.24	$2.1 \times 10^{-7}$	$2 \times 10^{-5}$ (chronic)	$6.9 \times 10^{-8}$	0.04	EPA IRIS	$2.7 \times 10^{-9}$ (extremely low)

source of data [ARCADIS 2009]

**ATSDR** = Agency for Toxic Substances and Disease Registry

**BaP TEq** = Benzo(a)Pyrene Toxicity Equivalents

**DBA** = Dibenzo(a,h)anthracene

**EPA IRIS** = U.S. Environmental Protection Agency Integrated Risk Information System [EPA 2013b]

**J** = estimated value

**mg/kg** = milligrams per kilogram

**mg/kg/day** = milligrams per kilogram per day

**NA** = non-applicable

**PAH** = polycyclic aromatic hydrocarbon

**PCB** = polychlorinated biphenyl

**µg/dL** = micrograms per deciliter

<sup>(1)</sup> = A relative bioavailability factor of 60% of the maximum concentration was used in the dose and risk calculations for arsenic [EPA 2015]

\*\* = Minimal risk levels for lead have not been established but the Centers for Disease Control and Prevention considers blood lead levels in children above 5µg/dL to be elevated

\*\*\* = The Centers for Disease Control and Prevention has not calculated a minimal risk level for PAHs but the maximum dose is well below the oral no adverse effect level of 1.3 mg/kg/day



**Table 7. Contaminant Concentrations in Off-Site Residential Surface Soil ( 0 to 6 inches deep) Near the Orlando Gasification Site - August 2006**

Contaminants	Sample Location <sup>(1)</sup> SS-29 (mg/kg)	Sample Location SS-30 (mg/kg)	Sample Location SS-31 (mg/kg)	Sample Location SS-32 (mg/kg)	Sample Location SS-33 (mg/kg)	Sample Location SS-34 (mg/kg)	Sample Location SS-35 (mg/kg)	Sample Location SS-36 (mg/kg)	Sample Location SS-37** (mg/kg)
Arsenic	0.35 U	0.5	0.2 U	0.8	0.4	0.4	0.3 U	0.2 U	13
DBA	0.18	0.01 U	0.01 U	0.01 U	0.01 U	0.01 U	0.01 U	0.19	0.01 U
Lead	75.3	136	53	58	162	25	51	44	485**
PAHs as B(a)P TEq	0.69	0.22	0.18	0.01	0.28	0.15	0.24	0.52	0.41
PCB	na	na	na	na	na	na	na	na	na

Data source [Arcadis 2009]

**B(a)P TEq** = Benzo(a)Pyrene Toxicity Equivalents

**CREG** = ATSDR cancer risk evaluation guide

**EPA** = US Environmental Protection Agency

**FDEP** = Florida Department of Environmental Protection

**mg/kg** = milligrams per kilogram

**PAH** = polycyclic aromatic hydrocarbon

**PCB** = polychlorinated biphenyl

**RMEG** = reference dose media evaluation guide

**SCTL** = soil cleanup target level

\* Screening guidelines only used to select chemicals for further scrutiny, not to judge the risk of illness.

\*\* In September 2015 the City of Orlando retested location SS-37 for lead. 5 surface soil samples were collected with results between 16 mg/kg and 75 mg/kg.

<sup>(1)</sup> sample locations are shown in Figure 4

**J** = estimated value

**na** = not analyzed for

**NA** = non-applicable

**U** = analyte not detected; reporting limit shown

**Table 7 (continued). Contaminant Concentrations in Off-Site Residential Surface Soil ( 0 to 6 inches deep) Near the Orlando Gasification Site - August 2006**

Contaminants	Sample Location ( <sup>1</sup> ) SS-38 (mg/kg)	Sample Location SS-39 (mg/kg)	Concentration Range (mg/kg)	Maximum Concentration in Surface Soil (mg/kg) [sample#]	Soil Screening Guideline (mg/kg)*	Source of Screening Guideline	# of samples above screening guideline/total # samples
Arsenic	0.6	0.2 U	0.2 U - 13	13 [SS-37]	15	RMEG	0/11
DBA	0.01 U	0.01 U	0.01 U - 0.19	0.19 [SS-36]	NA	NA	NA
Lead	38	49	25 - 485**	485 [SS-37]**	400	FDEP Residential SCTL	<b>1/11</b>
PAHs as B(a)P TEq	0.2	0.26	0.01 - 0.69	0.69 [SS-29]	0.1	CREG	<b>10/11</b>
PCB	Na	na	na	na	0.35	CREG	na

Data source [Arcadis 2009]

(<sup>1</sup>) sample locations are shown in Figure 4

**B(a)P TEq** = Benzo(a)Pyrene Toxicity Equivalents

**J** = estimated value

**CREG** = ATSDR cancer risk evaluation guide

**na** = not analyzed for

**EPA** = US Environmental Protection Agency

**NA** = non-applicable

**FDEP** = Florida Department of Environmental Protection

**U** = analyte not detected; reporting limit shown

**mg/kg** = milligrams per kilogram

**PAH** = polycyclic aromatic hydrocarbon

**PCB** = polychlorinated biphenyl

**RMEG** = reference dose media evaluation guide

**SCTL** = soil cleanup target level

\* Screening guidelines only used to select chemicals for further scrutiny, not to judge the risk of illness.

\*\* In September 2015 the City of Orlando retested location SS-37 for lead. 5 surface soil samples were collected with results between 16 mg/kg and 75 mg/kg.

**Table 8. Maximum Residential Soil Dose and Increased Cancer Risk From Inadvertent Soil Ingestion Near the Orlando Gasification Plant Site**

Contaminants	Maximum Off-Site Soil Concentration (0-6" deep) (mg/kg)	Estimated Maximum Residential Inadvertent Soil Ingestion Noncancer Dose (mg/kg/day)	ATSDR Minimal Risk Level (mg/kg/day)	Estimated Maximum Residential Inadvertent Soil Ingestion Cancer Dose (mg/kg/day)	Oral Cancer Slope Factor (mg/kg-day) <sup>-1</sup>	Source of Oral Cancer Slope Factor	Estimated Increased Cancer Risk
Arsenic	13 <sup>(1)</sup>	$1.4 \times 10^{-4}$	$3 \times 10^{-4}$ (chronic)	$4.1 \times 10^{-6}$	1.5	EPA IRIS	$3.1 \times 10^{-6}$ (extremely low)
DBA	0.51	$3.2 \times 10^{-7}$	none ***	$1.3 \times 10^{-7}$	7.3	EPA IRIS	$1.4 \times 10^{-5}$ (very low)
Lead	485	6.6µg/dL *	none **	NA	none	NA	NA
PAHs as BaP TEq	0.69	$4.3 \times 10^{-7}$	none ***	$1.8 \times 10^{-7}$	7.3	EPA IRIS	$2.0 \times 10^{-5}$ (extremely low)
PCB-1260	ND	NA	$2 \times 10^{-5}$ (chronic)	NA	0.04	EPA IRIS	NA

source of data [ARCADIS 2009]

**ATSDR** = Agency for Toxic Substances and Disease Registry

**BaP TEq** - Benzo(a)Pyrene Toxicity Equivalents

**DBA** = Dibenz(a,h)anthracene

**EPA IRIS** = U.S. Environmental Protection Agency Integrated Risk Information System [EPA 2013b]

**mg/kg** = milligrams per kilogram

**mg/kg/day** = milligrams per kilogram per day

**NA** = non-applicable

**PAH** = polycyclic aromatic hydrocarbon

**PCB** = polychlorinated biphenyl

**µg/dL** = micrograms per deciliter

<sup>(1)</sup> = A relative bioavailability factor of 60% of the maximum concentration was used in the dose and risk calculations for arsenic [EPA 2015]

\* = This is an estimate using EPA's IEUBK model of the blood lead level in children exposed to soil with a lead concentration of 485 mg/kg.

\*\* = Minimal risk levels for lead have not been established but the Centers for Disease Control and Prevention considers blood lead levels in children above 5µg/dL to be elevated

\*\*\* = The Centers for Disease Control and Prevention has not calculated a minimal risk level for carcinogenic PAHs but the maximum dose is well below the oral no adverse effect level of 1.3 mg/kg/day

**Table 9. Maximum Blood Lead Levels ( $\mu\text{g}/\text{dL}$ ) From Surface Soil On and Near the Orlando Gasification Plant Site**

<b>Age (Years)</b>	<b>On-Site Exposure at a Maximum Soil Lead Concentration of 80 mg/kg (April, 2004)</b>	<b>Residential Exposure at a Maximum Soil Lead Concentration of 75 mg/kg (Sept, 2015)</b>	<b>Off-Site Exposure Along West Robinson Street and Chatham Avenue at a Maximum Soil Lead Concentration of 110 mg/kg (April, 2004)</b>	<b>Off-Site Exposure Along the RR Track North of the Site at a Maximum Soil Lead Concentration of 1200 mg/kg (April, 2004)</b>
0.5-1	1.8	1.5	2.1	11.1
1-2	2.0	1.6	2.4	12.9
2-3	1.8	1.4	2.2	12.1
3-4	1.7	1.3	2.1	11.7
4-5	1.5	1.1	1.7	9.8
5-6	1.3	0.9	1.5	8.2
6-7	1.2	0.8	1.4	7.2

source of data [ARCADIS 2009]

**Note:** Blood lead levels were calculated using the EPA Integrated Exposure Uptake Biokinetic (IEUBK) model. It should be noted that there are uncertainties and limitations in the IEUBK model. One limitation is the inability to decrease the exposure frequency from 365 days per year. Another limitation is that it can only be used to calculate blood lead levels for children. Results above would be less if the exposure was less than 365 days per year or for adults.

$\mu\text{g}/\text{dL}$  = micrograms per deciliter

$\text{mg}/\text{kg}$  = milligrams per kilogram

**Table 10. Contaminant Concentrations in Off-Site Surface Soil (0-6 Inches Deep) Along West Robinson Street and Chatham Avenue at the Orlando Gasification Plant Site – April, 2004**

Contaminants	Concentration Range (mg/kg)	Maximum Concentration in Surface Soil (mg/kg) (sample#)	Soil Screening Guideline (mg/kg)*	Source of Screening Guideline	# of samples above screening guideline/total # samples
Arsenic	0.2 U - 1.2	1.2 (SS-9)	15	RMEG	0/7
DBA	0.02 – 0.37	0.37 (SS-7)	NA	NA	NA
Lead	11 J - 110 J	110 J (SS-9)	400	FDEP Residential SCTL	0/7
PAHs as BaP TEq	0.12 – 2.5	2.5 (SS-7)	0.1	CREG	<b>7/7</b>
PCB-1260	ND	NA	0.35	CREG	0/7

source of data [ARCADIS 2009]

**BaP TEq** = Benzo(a)Pyrene Toxicity Equivalents

**CREG** = ATSDR cancer risk evaluation guide

**DBA** = Dibenz(a,h)anthracene

**EPA** = U.S. Environmental Protection Agency

**FDEP** = Florida Department of Environmental Protection

**mg/kg** = milligrams per kilogram

**NA** = data either not analyzed or not available

**PAH** = polycyclic aromatic hydrocarbon

**PCB** = polychlorinated biphenyl

**RMEG** = reference dose media evaluation guide

**SCTL** = soil cleanup target level

**U** = analyte not detected; reporting limit shown

\* Screening guidelines only used to select chemicals for further scrutiny, not to judge the risk of illness.

**Table 11. Maximum Pedestrian Dose and Increased Cancer Risk from Inadvertent Ingestion of Off-Site Surface Soil Along West Robinson Street and Chatham Avenue Near the Orlando Gasification Plant Site**

Contaminants	Maximum Off-Site Soil Concentration (0-6" deep) (mg/kg)	Estimated Maximum Pedestrian Inadvertent Soil Ingestion Noncancer Dose (mg/kg/day)	ATSDR Minimal Risk Level (mg/kg/day)	Estimated Maximum Residential Inadvertent Soil Ingestion Cancer Dose (mg/kg/day)	Oral Cancer Slope Factor (mg/kg-day) <sup>-1</sup>	Source of Oral Cancer Slope Factor	Estimated Increased Cancer Risk
Arsenic	1.2 <sup>(1)</sup>	$5.2 \times 10^{-7}$	$3 \times 10^{-4}$ (chronic)	$1.3 \times 10^{-7}$	1.5	EPA IRIS	$2.0 \times 10^{-7}$ (extremely low)
DBA	0.37	$2.6 \times 10^{-7}$	none ***	$6.8 \times 10^{-8}$	7.3	EPA IRIS	$4.9 \times 10^{-7}$ (extremely low)
Lead	110 J	< 5µg/dL *	none **	NA	none	NA	NA
PAHs as BaP TEq	2.5	$1.8 \times 10^{-6}$	none ***	$4.6 \times 10^{-7}$	7.3	EPA IRIS	$3.4 \times 10^{-6}$ (extremely low)
PCB-1260	ND	NA	$2 \times 10^{-5}$ (chronic)	NA	0.04	EPA IRIS	NA

source of data [ARCADIS 2009]

**ATSDR** = Agency for Toxic Substances and Disease Registry

**BaP TEq** = Benzo(a)Pyrene Toxicity Equivalents

**DBA** = Dibenzo(a,h)anthracene

**EPA IRIS** = U.S. Environmental Protection Agency Integrated Risk Information System (EPA 2013b)

**J** = estimated value

**mg/kg** = milligrams per kilogram

**mg/kg/day** = milligrams per kilogram per day

**NA** = non-applicable

**PAH** = polycyclic aromatic hydrocarbon

**PCB** = polychlorinated biphenyl

**µg/dL** = micrograms per deciliter

<sup>(1)</sup> = A relative bioavailability factor of 60% of the maximum concentration was used in the dose and risk calculations for arsenic [EPA 2015]

\* = This is an estimate using EPA's IEUBK model of the blood lead level in children exposed to soil with a lead concentration of 110 mg/kg.

\*\* = Minimal risk levels for lead have not been established but the Centers for Disease Control and Prevention considers blood lead levels in children above 5µg/dL to be elevated

\*\*\* = The Centers for Disease Control and Prevention has not calculated a minimal risk level for PAHs but the maximum dose is well below the oral no adverse effect level of 1.3 mg/kg/day

**Table 12. Contaminant Concentrations in Surface Soil (0-6 Inches Deep) Along the Railroad Track North of the Orlando Gasification Plant Site – April, 2004**

Contaminants	Concentration Range (mg/kg)	Maximum Concentration in Surface Soil (mg/kg) (sample#)	Soil Screening Guideline (mg/kg)*	Source of Screening Guideline	# of samples above screening guideline/total # samples
Arsenic	6.9 (6.4)** - 39	39 (SS-19)	15	RMEG	<b>2/6</b>
DBA	1.1 – 6.7	6.7 (SS-20)	NA	NA	NA
Lead	230 J - 1200 J	1200 (SS-21)	400	FDEP Residential SCTL	<b>3/6</b>
PAHs as BaP TEq	7.9 – 65.4	65.4 (SS-20)	0.1	CREG	<b>6/6</b>
PCB-1260	NA	NA	0.35	CREG	0/6

source of data [ARCADIS 2009]

**BaP TEq** = Benzo(a)Pyrene Toxicity Equivalents

**CREG** = ATSDR cancer risk evaluation guide

**DBA** = Dibenzo(a,h)anthracene

**FDEP** = Florida Department of Environmental Protection

**J** = estimated value

**mg/kg** = milligrams per kilogram

**NA** = data either not analyzed or not available

**PAH** = polycyclic aromatic hydrocarbon

**PCB** = polychlorinated biphenyl

**RMEG** = reference dose media evaluation guide

**SCTL** = soil cleanup target level

\* Screening guidelines only used to select chemicals for further scrutiny, not to judge the risk of illness.

**Table 13. Maximum Trespasser Dose and Increased Risk From Inadvertent Ingestion of Surface Soil (0-6 Inches Deep) Along the RR Track North of the Orlando Gasification Plant Site**

<b>Contaminants</b>	<b>Maximum Off-Site Soil Concentration (0-6" deep) (mg/kg)</b>	<b>Estimated Maximum Trespasser Inadvertent Soil Ingestion Dose (mg/kg/day)</b>	<b>ATSDR Minimal Risk Level (mg/kg/day)</b>
Arsenic	39 <sup>(1)</sup>	$3.0 \times 10^{-5}$	$3 \times 10^{-4}$ (chronic)
DBA	6.7	NC	NA
Lead	1200	< 5µg/dL *	none**
PAHs (Noncarcinogenic)	140	NC	NA
PCB-1260	ND	NA	$2 \times 10^{-5}$ (chronic)

Source of data [ARCADIS 2009]

**ATSDR** = Agency for Toxic Substances and Disease Registry

**DBA** = Dibenzo(a,h)anthracene

**mg/kg** = milligrams per kilogram

**mg/kg/day** = milligrams per kilogram per day

**NA** = non-applicable

**NC** = Dose was not calculated since noncarcinogenic PAH concentrations were below EPA screening levels

**ND** = below detection level

**PAH** = polycyclic aromatic hydrocarbon

**PCB** = polychlorinated biphenyl

**µg/dL** = micrograms per deciliter

<sup>(1)</sup> = A relative bioavailability factor of 60% of the maximum concentration was used in the dose and risk calculations for arsenic [EPA 2015]

\* = This is an estimate using EPA's IEUBK model of the blood lead level in children exposed to soil with a lead concentration of 1200 mg/kg.

\*\* = Minimal risk levels for lead have not been established but the Centers for Disease Control and Prevention considers blood lead levels in children above 5µg/dL to be elevated



## **Appendix C**

### **Figures**



MAP SOURCE: USGS TOPOGRAPHIC QUADRANGLE  
ORLANDO, FLORIDA

0 1000 2000  
SCALE IN FEET

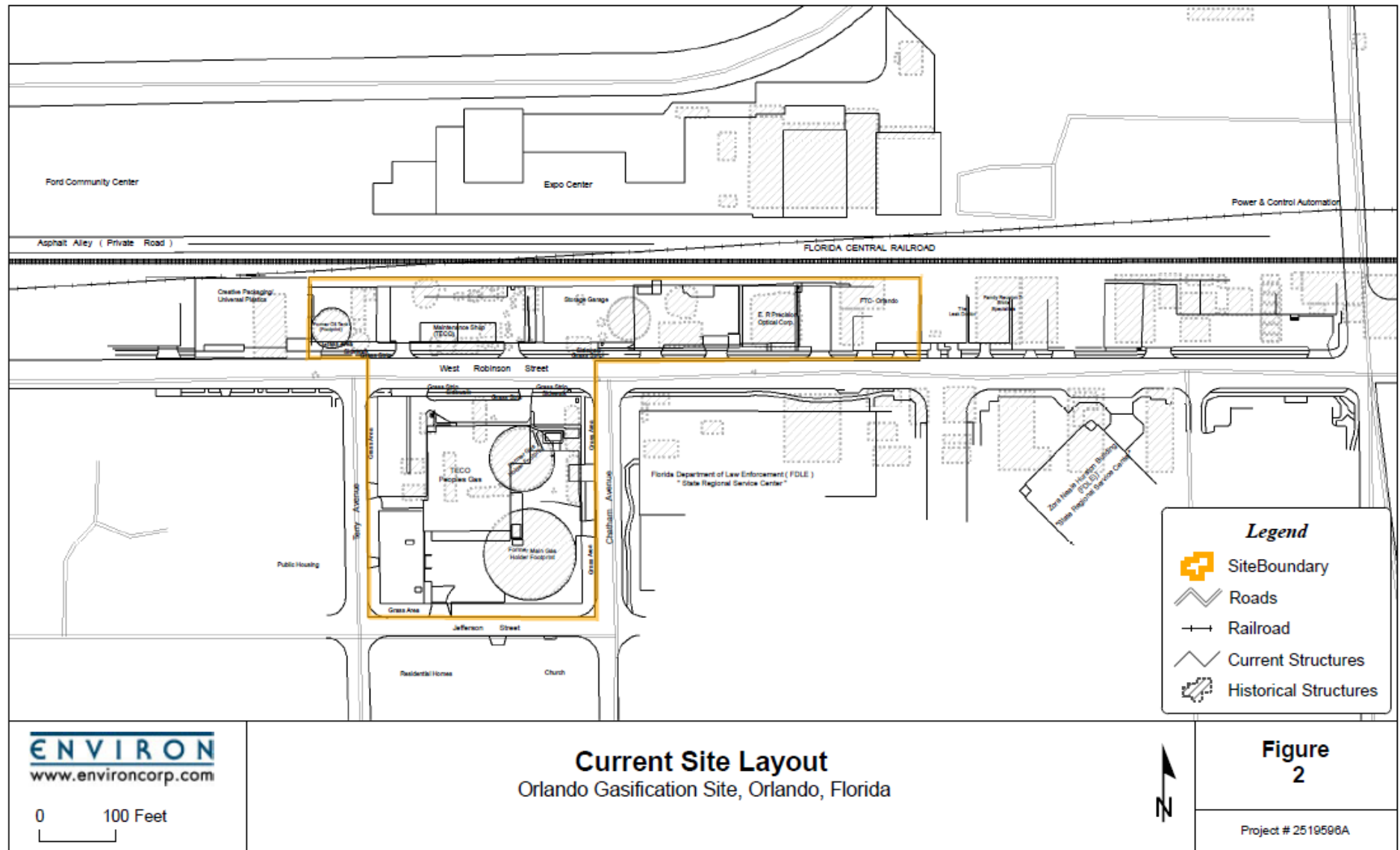
ORLANDO GASIFICATION SITE  
600 W. ROBINSON ST., ORLANDO, FLORIDA

SITE LOCATION MAP

**ARCADIS**

FIGURE 1

### Figure 2. Orlando Gasification Current Site Layout



**Figure 3. Orlando Gasification On-Site Surface Features**

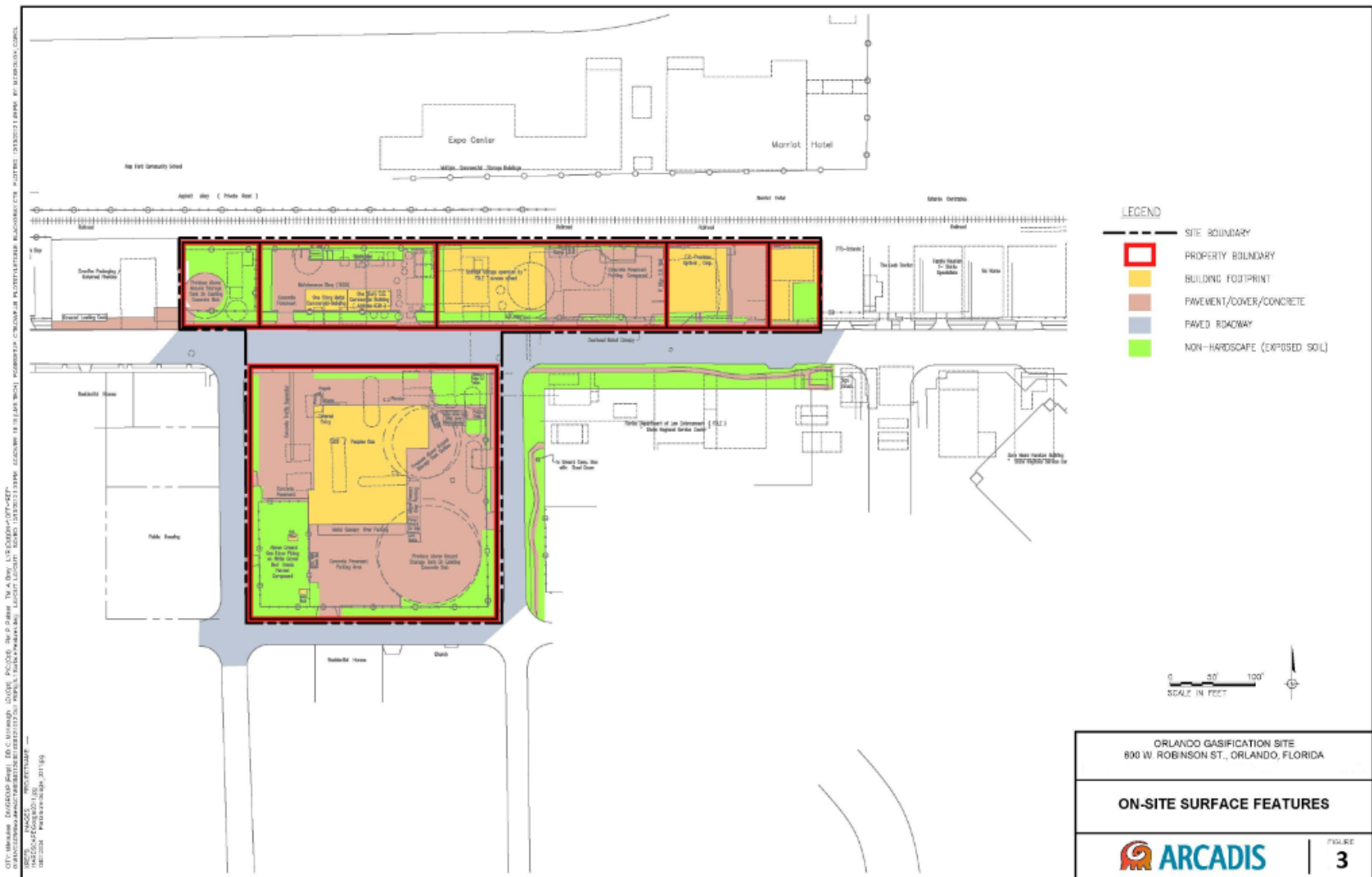
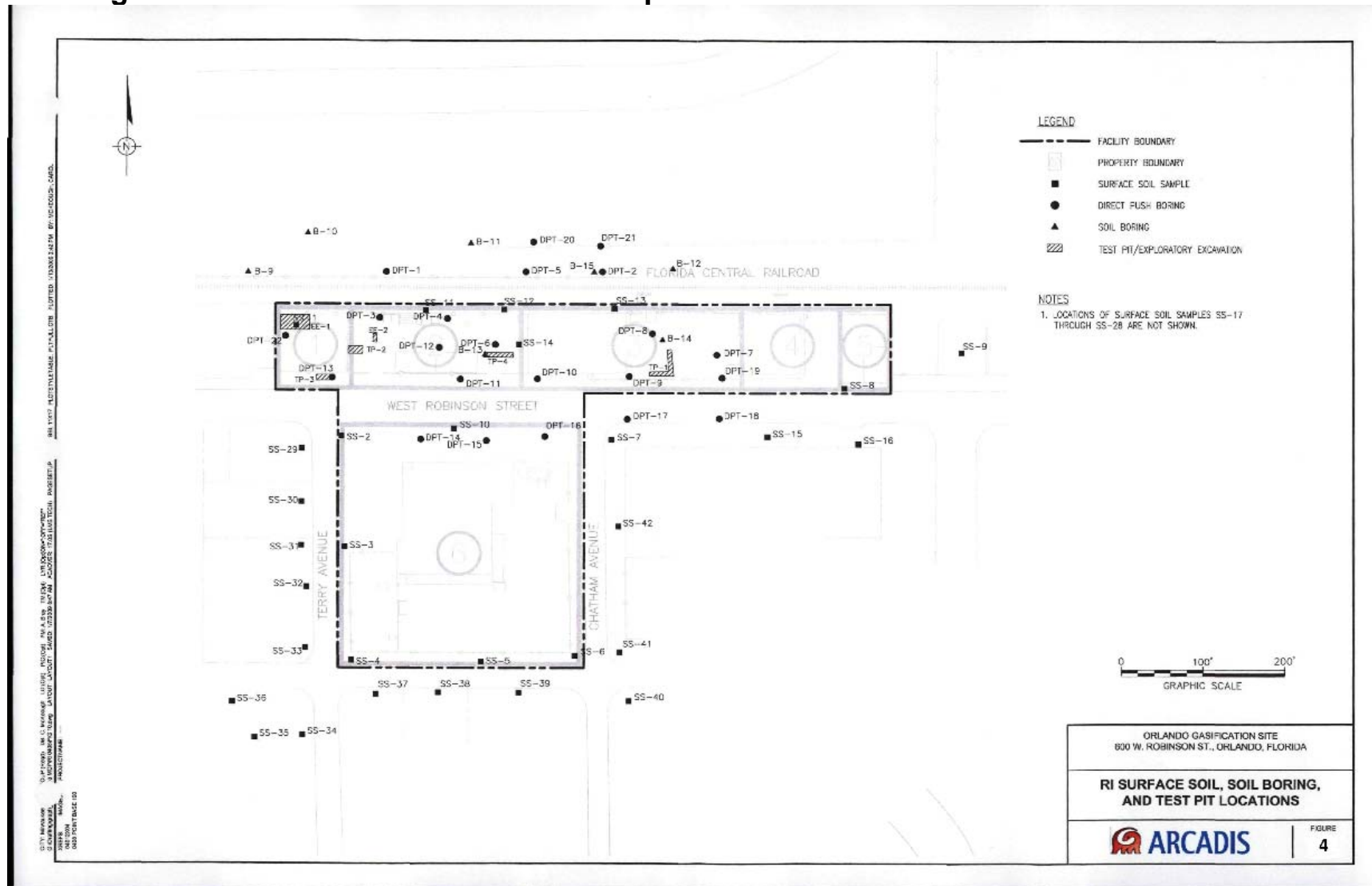
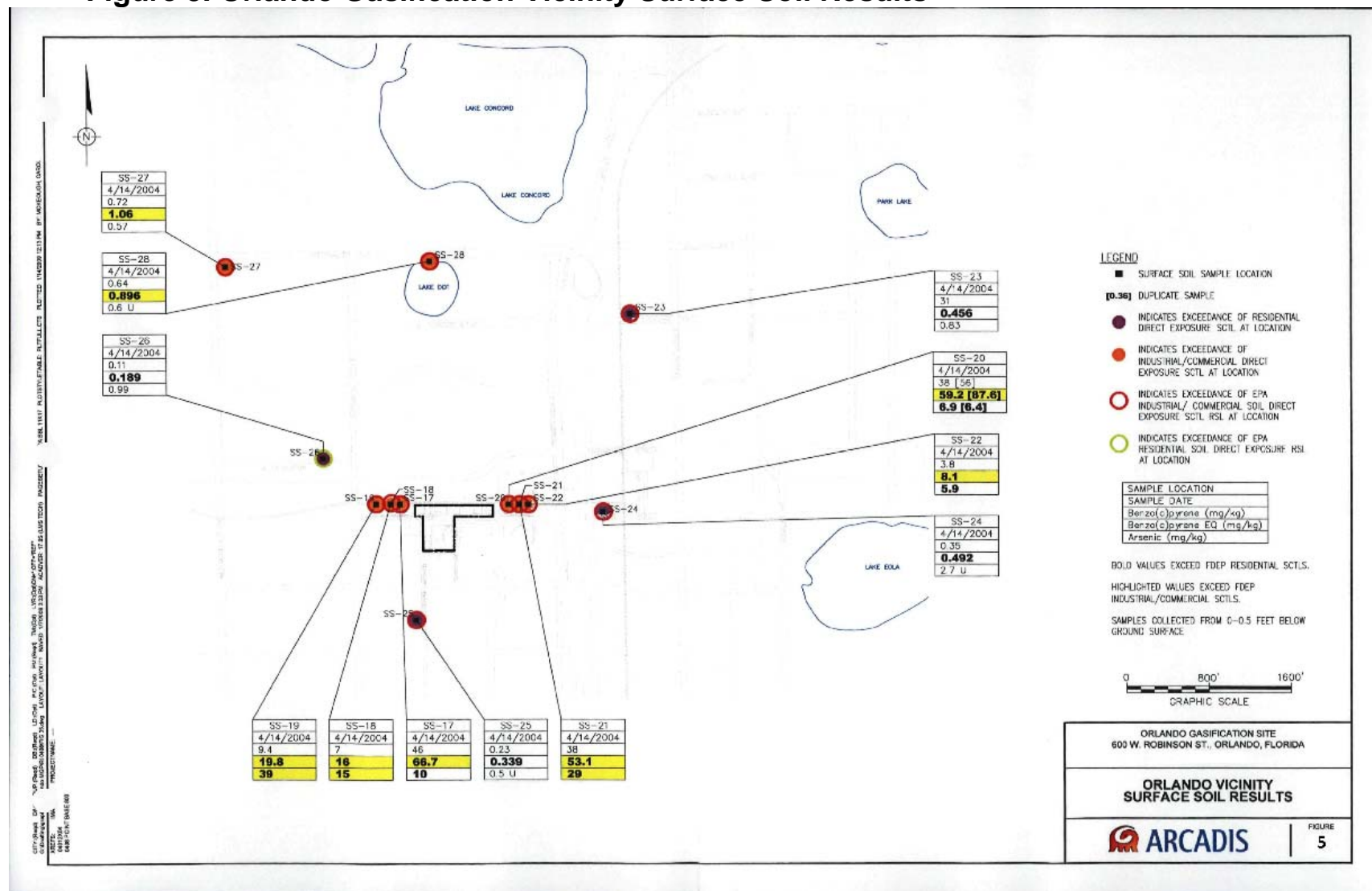


Figure 4. Orlando Gasification Soil Sample Locations

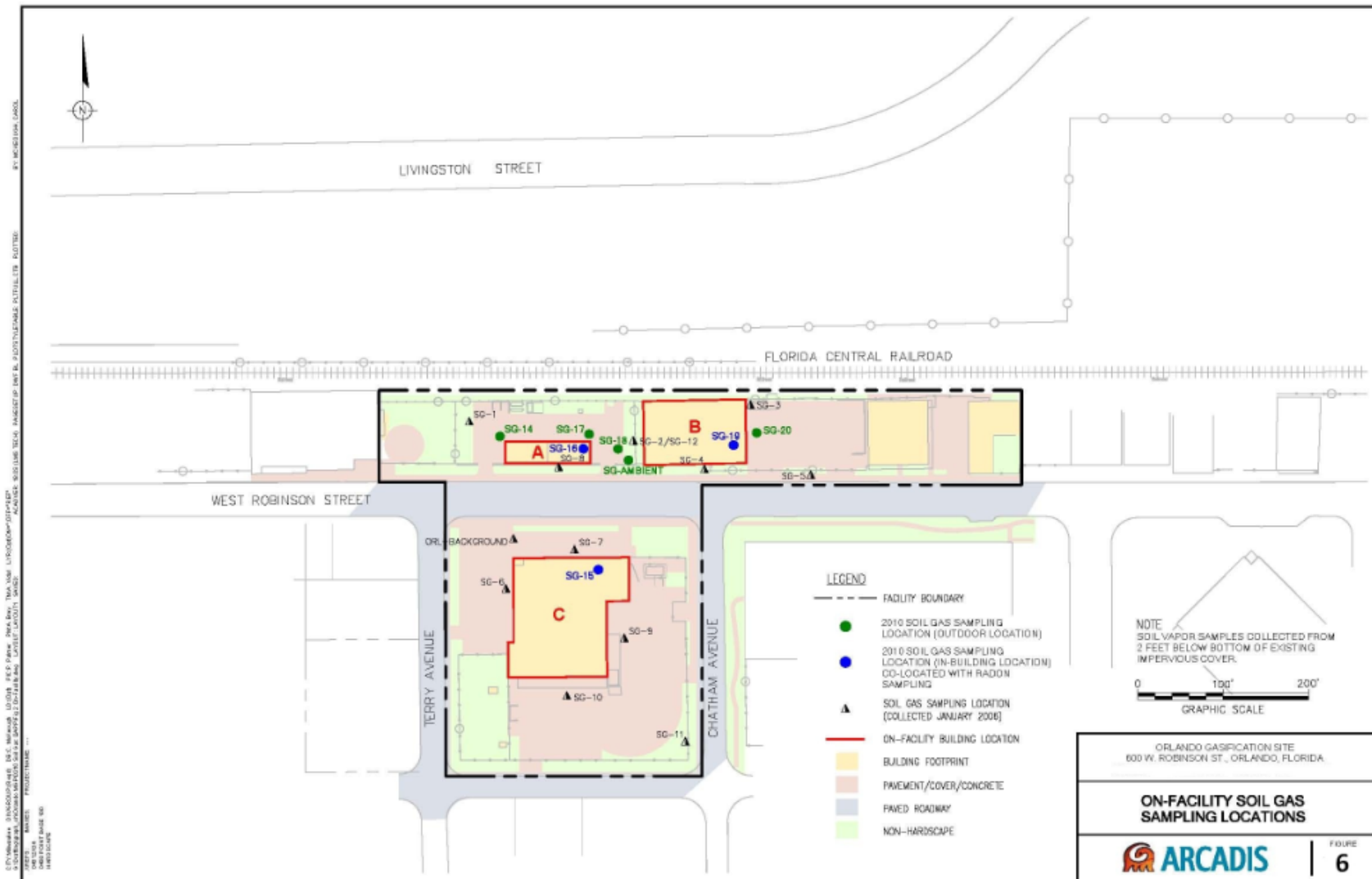




### Figure 5. Orlando Gasification Vicinity Surface Soil Results



**Figure 6. Orlando Gasification On-Facility Soil Gas Sampling Locations**





## **Appendix D**

***Office of Environmental Health Hazard Assessment (OEHHA)***

***Potency Equivalency Factors (PEFs)***

***For PAHs based on Benzo(a)pyrene Potency***



**OEHHA PEF WEIGHTING SCHEME FOR PAHS AND THEIR RESULTING CANCER POTENCY VALUES**

<b>PAH or derivative</b>	<b>PEF</b>	<b>Unit Risk (<math>\mu\text{g}/\text{m}^3</math>)<sup>-1</sup></b>	<b>Inhalation Slope Factor (<math>\text{mg}/\text{kg}\cdot\text{day}</math>)<sup>-1</sup></b>	<b>Oral Slope Factor (<math>\text{mg}/\text{kg}\cdot\text{day}</math>)<sup>-1</sup></b>
<b>benzo[a]pyrene</b> (index compound)	<b>1.0</b>	<b><math>1.1 \times 10^{-3}</math></b>	<b>3.9</b>	<b><math>1.2 \times 10^{+1}</math></b>
benz[a]anthracene	0.1	$1.1 \times 10^{-4}$	$3.9 \times 10^{-1}$	1.2
benzo[b]fluoranthene	0.1	$1.1 \times 10^{-4}$	$3.9 \times 10^{-1}$	1.2
benzo[j]fluoranthene	0.1	$1.1 \times 10^{-4}$	$3.9 \times 10^{-1}$	1.2
benzo[k]fluoranthene	0.1	$1.1 \times 10^{-4}$	$3.9 \times 10^{-1}$	1.2
dibenz[a,j]acridine	0.1	$1.1 \times 10^{-4}$	$3.9 \times 10^{-1}$	1.2
dibenz[a,h]acridine	0.1	$1.1 \times 10^{-4}$	$3.9 \times 10^{-1}$	1.2
7H-dibenzo[c,g]carbazole	1.0	$1.1 \times 10^{-3}$	3.9	$1.2 \times 10^{+1}$
dibenzo[a,e]pyrene	1.0	$1.1 \times 10^{-3}$	3.9	$1.2 \times 10^{+1}$
dibenzo[a,h]pyrene	10	$1.1 \times 10^{-2}$	$3.9 \times 10^{+1}$	$1.2 \times 10^{+2}$
dibenzo[a,i]pyrene	10	$1.1 \times 10^{-2}$	$3.9 \times 10^{+1}$	$1.2 \times 10^{+2}$
dibenzo[a,l]pyrene	10	$1.1 \times 10^{-2}$	$3.9 \times 10^{+1}$	$1.2 \times 10^{+2}$
indeno[1,2,3-cd]pyrene	0.1	$1.1 \times 10^{-4}$	$3.9 \times 10^{-1}$	1.2
5-methylchrysene	1.0	$1.1 \times 10^{-3}$	3.9	$1.2 \times 10^{+1}$
1-nitropyrene	0.1	$1.1 \times 10^{-4}$	$3.9 \times 10^{-1}$	1.2
4-nitropyrene	0.1	$1.1 \times 10^{-4}$	$3.9 \times 10^{-1}$	1.2
1,6-dinitropyrene	10	$1.1 \times 10^{-2}$	$3.9 \times 10^{+1}$	$1.2 \times 10^{+2}$
1,8-dinitropyrene	1.0	$1.1 \times 10^{-3}$	3.9	$1.2 \times 10^{+1}$
6-nitrochrysene	10	$1.1 \times 10^{-2}$	$3.9 \times 10^{+1}$	$1.2 \times 10^{+2}$
2-nitrofluorene	0.01	$1.1 \times 10^{-5}$	$3.9 \times 10^{-2}$	$1.2 \times 10^{-1}$
chrysene	0.01	$1.1 \times 10^{-5}$	$3.9 \times 10^{-2}$	$1.2 \times 10^{-1}$

Source: [OEHHA 1993]



## Glossary

### **Absorption**

The process of taking in. For a person or animal, absorption is the process of a substance getting into the body through the eyes, skin, stomach, intestines, or lungs.

### **Acute**

Occurring over a short time (compare with **chronic**).

### **Acute exposure**

Contact with a substance that occurs once or for only a short time (up to 14 days) (compare with **intermediate duration exposure** and **chronic exposure**).

### **Adverse health effect**

A change in body function or cell structure that might lead to disease or health problems.

### **Ambient**

Surrounding (for example, *ambient* air).

### **Analyte**

A substance measured in the laboratory. A chemical for which a sample (such as water, air, or blood) is tested in a laboratory. For example, if the analyte is mercury, the laboratory test will determine the amount of mercury in the sample.

### **Cancer**

Any one of a group of diseases that occurs when cells in the body become abnormal and grow or multiply out of control.

### **Cancer risk**

A theoretical risk of for getting cancer if exposed to a substance every day for 70 years (a lifetime exposure). The true risk might be lower.

### **Carcinogen**

A substance that causes cancer.

### **Chronic**

Occurring over a long time (more than 1 year) (compare with **acute**).

### **Chronic exposure**

Contact with a substance that occurs over a long time (more than 1 year) (compare with **acute exposure** and **intermediate duration exposure**).

**Comparison value (CV)**

Calculated concentration of a substance in air, water, food, or soil that is unlikely to cause harmful (adverse) health effects in exposed people. The CV is used as a screening level during the public health assessment process. Substances found in amounts greater than their CVs might be selected for further evaluation in the public health assessment process.

**Completed exposure pathway** (see **exposure pathway**).

**Concentration**

The amount of a substance present in a certain amount of soil, water, air, food, blood, hair, urine, breath, or any other media.

**Contaminant**

A substance that is either present in an environment where it does not belong or is present at levels that might cause harmful (adverse) health effects.

**Dermal**

Referring to the skin. For example, dermal absorption means passing through the skin.

**Dermal contact**

Contact with (touching) the skin (see **route of exposure**).

**Detection levels**

The lowest concentration of a chemical that can reliably be distinguished from a zero concentration.

**Dose (for chemicals that are not radioactive)**

The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An “exposure dose” is how much of a substance is encountered in the environment. An “absorbed dose” is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines, or lungs.

**Environmental media**

Soil, water, air, biota (plants and animals), or any other parts of the environment that can contain contaminants.

**Environmental media and transport mechanism**

Environmental media include water, air, soil, and **biota** (plants and animals). Transport mechanisms move contaminants from the source to points where human exposure can occur. The **environmental media and transport mechanism** is the second part of an **exposure pathway**.

**EPA**

United States Environmental Protection Agency.

**Epidemiology**

The study of the distribution and determinants of disease or health status in a population; the study of the occurrence and causes of health effects in humans.

**Exposure**

Contact with a substance by swallowing, breathing, or touching the skin or eyes. Exposure may be short-term (**acute exposure**), of intermediate duration, or long-term (**chronic exposure**).

**Exposure assessment**

The process of finding out how people come into contact with a hazardous substance, how often and for how long they are in contact with the substance, and how much of the substance they are in contact with.

**Exposure pathway**

The route a substance takes from its source (where it began) to its end point (where it ends), and how people can come into contact with (or get exposed to) it. An exposure pathway has five parts: a source of contamination (such as an abandoned business); an environmental media and transport mechanism (such as movement through groundwater); a point of exposure (such as a private well); a route of exposure (eating, drinking, breathing, or touching), and a receptor population (people potentially or actually exposed). When all five parts are present, the exposure pathway is termed a completed exposure pathway.

**Groundwater**

Water beneath the earth's surface in the spaces between soil particles and between rock surfaces (compare with **surface water**).

**Hazard**

A source of potential harm from past, current, or future exposures.

**Hazardous waste**

Potentially harmful substances that have been released or discarded into the environment.

**Health consultation**

A review of available information or collection of new data to respond to a specific health question or request for information about a potential environmental hazard. Health consultations are focused on a specific exposure issue. Health consultations are therefore more limited than a public health assessment, which reviews the exposure potential of each pathway and chemical (compare with **public health assessment**).

**Ingestion**

The act of swallowing something through eating, drinking, or mouthing objects. A hazardous substance can enter the body this way (see **route of exposure**).

**Inhalation**

The act of breathing. A hazardous substance can enter the body this way (see **route of exposure**).

**Intermediate duration exposure**

Contact with a substance that occurs for more than 14 days and less than a year (compare with **acute exposure** and **chronic exposure**).

**Lowest-observed-adverse-effect level (LOAEL)**

The lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects in people or animals.

**mg/kg**

Milligram per kilogram.

**Minimal risk level (MRL)**

An ATSDR estimate of daily human exposure to a hazardous substance at or below which that substance is unlikely to pose a measurable risk of harmful (adverse), noncancerous effects. MRLs are calculated for a route of exposure (inhalation or oral) over a specified time period (acute, intermediate, or chronic). MRLs should not be used as predictors of harmful (adverse) health effects (see **reference dose**).

**No-observed-adverse-effect level (NOAEL)**

The highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals.

**Plume**

A volume of a substance that moves from its source to places farther away from the source. Plumes can be described by the volume of air or water they occupy and the direction they move. For example, a plume can be a column of smoke from a chimney or a substance moving with groundwater.

**Point of exposure**

The place where someone can come into contact with a substance present in the environment (see **exposure pathway**).

**Population**

A group or number of people living within a specified area or sharing similar characteristics (such as occupation or age).

**Potentially responsible party (PRP)**

A company, government, or person legally responsible for cleaning up the pollution at a hazardous waste site under Superfund. There may be more than one PRP for a particular site.



**Prevention**

Actions that reduce exposure or other risks, keep people from getting sick, or keep disease from getting worse.

**Public comment period**

An opportunity for the public to comment on agency findings or proposed activities contained in draft reports or documents. The public comment period is a limited time period during which comments will be accepted.

**Public health assessment (PHA)**

An ATSDR document that examines hazardous substances, health outcomes, and community concerns at a hazardous waste site to determine whether people could be harmed from coming into contact with those substances. The PHA also lists actions that need to be taken to protect public health (compare with **health consultation**).

**Public meeting**

A public forum with community members for communication about a site.

**Receptor population**

People who could come into contact with hazardous substances (see **exposure pathway**).

**Reference dose (RfD)**

An EPA estimate, with uncertainty or safety factors built in, of the daily lifetime dose of a substance that is unlikely to cause harm in humans.

**Registry**

A systematic collection of information on persons exposed to a specific substance or having specific diseases (see **exposure registry** and **disease registry**).

**Remedial Investigation**

The CERCLA process of determining the type and extent of hazardous material contamination at a site.

**RfD**

See **reference dose**.

**Risk**

The probability that something will cause injury or harm.

**Route of exposure**

The way people come into contact with a hazardous substance. Three routes of exposure are breathing (**inhalation**), eating or drinking (**ingestion**), or contact with the skin (**dermal contact**).

**Sample**

A portion or piece of a whole. A selected subset of a population or subset of whatever is being studied. For example, in a study of people the sample is a number of people chosen from a larger

population (see **population**). An environmental sample (for example, a small amount of soil or water) might be collected to measure contamination in the environment at a specific location.

**Solvent**

A liquid capable of dissolving or dispersing another substance (for example, acetone or mineral spirits).

**Source of contamination**

The place where a hazardous substance comes from, such as a landfill, waste pond, incinerator, storage tank, or drum. A source of contamination is the first part of an **exposure pathway**.

**Substance**

A chemical.

**Surface water**

Water on the surface of the earth, such as in lakes, rivers, streams, ponds, and springs (compare with **groundwater**).

**Toxicological profile**

An ATSDR document that examines, summarizes, and interprets information about a hazardous substance to determine harmful levels of exposure and associated health effects. A toxicological profile also identifies significant gaps in knowledge on the substance and describes areas where further research is needed.

**Toxicology**

The study of the harmful effects of substances on humans or animals.

**Uncertainty factor**

Mathematical adjustments for reasons of safety when knowledge is incomplete. For example, factors used in the calculation of doses that are not harmful (adverse) to people. These factors are applied to the lowest-observed-adverse-effect-level (LOAEL) or the no-observed-adverse-effect-level (NOAEL) to derive a minimal risk level (MRL). Uncertainty factors are used to account for variations in people's sensitivity, for differences between animals and humans, and for differences between a LOAEL and a NOAEL. Scientists use uncertainty factors when they have some, but not all, the information from animal or human studies to decide whether an exposure will cause harm to people (also sometimes called a **safety factor**).

**Volatile organic compounds (VOCs)**

Organic compounds that evaporate readily into the air. VOCs include substances such as benzene, toluene, methylene chloride, and methyl chloroform.